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THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey
BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING
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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station — Beltsville, Maryland
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RECENT EPIDEMICS OF DOWNY MILDEW OF ROSE

Kenneth F. Baker[1]

Serious epidemics of downy mildew (Peronospora sparsa Berk.) of cultivated roses occurred in the San Francisco Bay region of California during the fall and winter of 1950-51 (29, 56), and in Richmond, Indiana during July and August, 1951. This was the first reappearance of the disease in California since that observed by Dr. J. T. Barrett in 1929 in the same area. The Indiana outbreak may be the first record in that State, but the fungus had been collected in nearby Ohio in 1905 (Cincinnati, November 1, by J. A. Peterson, Mycological Collections of the Bureau of Plant Industry) and perhaps in Iowa in 1914 (19, 41).

Because of the highly sporadic occurrence of this fungus, the more than 120[2] papers concerning it are widely scattered in the literature and pathologists frequently underestimate the potential importance of the disease it causes. It has seemed desirable, therefore, to present briefly for this disease the history, occurrence, predisposing environmental conditions, and recommended control procedures.

History and Occurrence

The disease was first reported by Berkeley (5) in 1862 as causing considerable damage to roses in a cool glasshouse in England. It occurred in Italy in 1875 (C. Bagnis, Micologia Romana, Century I, p. 19. Atti Reale Accad. Lincei 274. 1876-77; cited in 7, 12). Ritzema Bos (44) states that it was present in Germany in 1870, but the first definite report found is that of Wittmack (55), who found the disease near Berlin in 1876. In the next few years it was very damaging to greenhouse roses there. Since that time the disease has been found in most of the countries of Europe. It was reported in Austria and France in 1880 (12); in Denmark in 1884 (45) when it killed 2000 bushes; in Silesia in 1891 (49), where it killed 400,000 bushes; in the Russian Caucasus in 1897 (50); in Holland in 1901 and 1902 (44); in Sweden in 1905 (Eriksson, Meddel. Kungl. Landtbruks Akad. Exper. No. 92, 1906; cited in 42); in Russia near Moscow and Leningrad in 1923 (26); in Egypt in 1924-25 (15), in Poland in 1926 (17, 26); in Norway in 1927 (26); in Roumania in 1932, as Peronospora rosae gallicae Sav. & Rayss (46); in Czechoslovakia by 1934 (46); in British Columbia, Canada in 1937 (25); in Brazil in 1939 (35); in Switzerland in 1940 (30); and in Iceland in 1950 (13).

In the United States, the disease was first reported in 1880 by Harkness and Moore (24) near San Rafael, California on Rosa californica Cham. & Schlecht. It was subsequently observed on glasshouse roses by Plunkett (38) in southern California in the fall of 1927, and by Dr. J. T. Barrett in the San Francisco area in the fall of 1929. Besides the 1950-51 reappearance in the latter area, the fungus was again collected in a San Pablo glasshouse on the Red Delight variety in August-September, 1952 by Dr. M. W. Gardner and Dr. R. D. Raabe.

The fungus was reported in Geneva, New York in 1891 by Fairchild, as causing severe defoliation and losses (51), and was again collected there the following year (G. A. Atwood, May 1892, Mycological Collections of the Bureau of Plant Industry). In Pennsylvania, it was collected in Philadelphia in 1892 (Ellis, North American Fungi 1415). Ricker (43) found the fungus on greenhouse roses in Bangor, Maine in April 1900. In Ohio, as already mentioned, it appeared in November 1905. In New Jersey, Peronospora sparsa was definitely reported in 1912 (11); the fungus was mentioned in an 1893 report of Halsted (23), but it is doubtful whether the paper was prompted by the occurrence of the fungus there. It was reported by F. H. Blodgett (files of the Plant Disease Survey) in four counties in Texas in May-June, 1914. The fungus was reported in Iowa by Raeder (41) in 1914, but later examination (19) of one of his specimens revealed no downy mildew. In Idaho the parasite was collected by Willis in 1915 (47). In Florida it was present by 1942 (53, and perhaps 48).

It is of interest that all of the world distribution records are from north of the Tropic of Cancer except that from São Paulo, Brazil which lies on a cool plateau just north of the Tropic of Capricorn. The wild species of the genus Rosa are also distributed in the Northern Hemisphere, and it is highly probable that the fungus originated on them there. However, examina-

[1]Dr. P. R. Miller and the late Dr. W. L. White kindly made available references listed in the Division of Mycology and Disease Survey and the Farlow Herbarium, respectively. The author is indebted to Dr. Neil A. MacLean for assistance in some aspects of the work herein presented.

[2]These papers appeared rather regularly from 1860 to 1951 except during the periods of World Wars I and II.

tion of the distribution data affords no further clue as to where or when this pathogen originated. It was generally distributed over North America and Europe by the time mycologists began collecting fungi. The roses grown before 1850 were either wild species or not far removed from them. With increased breeding of roses following 1850 there was considerable international exchange of plant material (2), and it would appear probable that the fungus soon became widely distributed through shipment of infected cuttings and seedlings. The severe damage reported at the time of its first appearance in 1862 suggests that it had been present in England prior to that time.

Rostrup (45) suggested in 1902 or earlier that the organism originated in India, presumably because he found it as a parasite on Rosa indica Lindl. There is no evidence to substantiate this idea, for apparently the fungus is unknown in India. It is not reported there by Butler and Bisby (10), nor in the list prepared in 1950 by the Plant Pathology Committee, Indian Council of Agricultural Research (37), in spite of the fact that it is given for England in both the 1934 and 1944 lists of the British Mycological Society, on which the Indian tabulation was based. Furthermore, Dr. G. R. Bisby has written that there are no records or specimens of the fungus for India in the Commonwealth Mycological Institute. Despite these facts, several authors (e.g., 14, 20, 28, 35) have perpetuated Rostrup's misconception. Cuboni (12) thought the fungus originated in North America, but there is no more reason to believe that it originated here than in Europe.

Environmental Relations

Apparently the fungus has occurred as sporadically elsewhere as in California. Since its first report in England in 1862, it has attracted notice in 1910, 1923, 1928, 1934, and 1939 (8, 21, 22, 31). In Sweden it was severe in 1905, 1910, and 1912 (22, 42); in Italy in 1875, 1888, and 1937 (7, 12, 40); in Germany in 1876, 1877, 1879, 1891, 1924, and 1928 (12, 27, 32, 49, 55); in Holland in 1901, 1902, and 1946 (4, 44); in Poland in 1926, 1929, and 1930 (17, 26); and in France in 1880 and 1916-17 (12, 39). The recurrence of the disease at long intervals presupposes an effective means of carryover of the fungus. An extraordinary epidemic of downy mildews on many genera of wild annuals observed in the arid deserts of the Imperial and Coachella Valleys of southern California during the wet winter of 1940-41 indicated that such prolonged carryover is possible in the downy mildews.

Since most of the sporadic outbreaks took place in the controlled environment of glasshouses and occurrence is relatively rare outdoors (28, 34), it would appear that the fungus has narrow temperature and humidity requirements. This rarity might also be due to the fact that infection of rose leaves and stems is largely restricted to young apical growth, in a zone with lower humidity than prevails at the soil surface. An outbreak could occur, then, on the young leaves of small plants under moderately moist conditions, whereas protracted very wet conditions would be required for an epidemic on mature plants. A parallel situation has been observed with downy mildew of snapdragon (Peronospora antirrhini Schroet.) in California during the last 14 years. This fungus occurs practically every year on young seedlings in the humid San Francisco Bay area, and, under particularly moist conditions, may attack the growing points of mature plants two feet or more above the soil. On the other hand, snapdragon seedlings are infected by downy mildew under only especially moist shaded conditions in semiarid southern California, and mature plants have not been observed to be infected there.

The temperature and humidity relationships of the rose fungus reported elsewhere (29) are summarized here. Conidia of P. sparsa did not germinate at 40°F., but their viability was not reduced by such temperatures. The optimum for germination was 65° F., with a slight decrease at 70°, and no germination at 80°. When temperatures of 80° were maintained for 24 hours the conidia were killed. One grower successfully controlled the disease in his range by lowering the humidity and raising the temperature to 80° during the warmer part of the day.

There are no precise data on the effect of humidity on development of the fungus. Observations, however, indicate a positive correlation between the incidence of disease and relative humidity (29). In general, glasshouse crops "grown wet" (walks and beds very wet, ventilators closed, minimal heat used) had severe downy mildew, whereas adjacent crops "grown dry" (walks not soaked, ventilators open, heat used to reduce humidity) had no trouble with the disease. Similar but smaller variations sometimes occurred within one glasshouse owing to roof leaks, poor heat distribution, etc.

Tests were conducted on the control of the disease by decreasing the humidity in glasshouses. Heat was used mostly to maintain a safe humidity, rather than for temperature control as usually practiced. The proper balancing of heat and ventilation in glasshouses permits ef-

fective humidity control (3), particularly when operation is based on readings of humidity indicators. With lowered humidity and subsequent drying of plant surfaces there was a corresponding decrease in the amount of downy mildew. When humidity was kept below 85 percent the roses remained uninfected and developed vigorous new growth.

Recent Epidemics in the United States

The 1950-51 California Outbreak: The 1950-51 outbreak in the San Leandro and Richmond areas of central California apparently was more destructive than most previously reported epidemics. Very few flowers for the Christmas market were cut in glasshouses where the epidemic occurred, and several large glasshouse ranges were involved. Losses in one large range were offset to some extent by rapid recovery of plants and production of a good crop following control of the disease by humidity reduction. Precipitation in the last three months of 1950 in the San Francisco Bay area was well above average[3], whereas in the early months of 1951 it was below normal (Table 1). The weather data for the San Leandro area are represented by the nearby Hayward, Oakland, and Oakland Airport stations, that of the Richmond area is supplemented by data from adjacent Berkeley. November rainfall undoubtedly was largely responsible for the outbreak. For example, Hayward reported 11.22 inches in eight days, with 7.15 inches of it on November 18. The latter deluge flooded one rose range at San Lorenzo with nearly two feet of water and put the heating plant out of operation. In the Los Angeles area, on the other hand, the deficient rainfall precluded downy mildews generally. Crops on the San Francisco peninsula likewise escaped, apparently because of proper humidity control, since rainfall and humidity (see Redwood City data) were high. The relative humidity in the Bay area was generally high during the winter of 1950-51. Because temperatures did not reach low levels many greenhouses were not heated and therefore remained humid. There was a deficiency of sunny weather, glasshouses did not dry out, and conditions were favorable for growth, sporulation, and infection by downy mildew. In contrast, midwestern and eastern greenhouses have low humidity in winter months and should not have trouble at those times.

Table 1. Rainfall at several locations in California during the wettest months of 1950-51.

Location	Period	Inches of rain in month of							
		Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	March	April
Hayward	1950-51	.10	2.45	11.56	7.46	4.46	2.93	4.16	1.07
Oakland Airport	1950-51	.01	1.80	4.64	5.36	4.34	2.46	1.46	.95
	Average	.41	.93	2.06	3.46	3.58	3.24	2.56	1.52
Oakland	1950-51	.01	2.93	6.14	7.51	6.50	3.06	1.87	1.21
	Average	.39	1.27	2.59	4.09	4.92	4.20	3.50	1.62
Berkeley	1950-51	0	3.28	7.42	6.67	5.03	2.58	1.53	1.24
	Average	.42	1.22	1.86	4.15	5.08	4.48	3.62	1.53
Richmond	1950-51	--	--	--	7.53	4.56	2.53	1.08	.86
Redwood City	1950-51	Trace	1.66	6.54	5.69	2.92	2.21	1.18	.89
	Average	.08	.94	1.44	3.84	4.71	4.31	2.80	1.30
Los Angeles	1950-51	.38	.24	1.05	.03	2.80	1.48	.44	1.54
	Average	.17	.68	1.20	2.63	3.10	3.07	2.78	1.04

[3]Weather data in this paper are taken from Climatological Data, California and Indiana Sections, of the U. S. Weather Bureau.

The heavy rainfall in the San Francisco Bay area in 1950-51 apparently was insufficient for an outbreak of the disease on outdoor roses. No complaints on this type of occurrence have come to our attention despite the rather general interest in the epidemic under glass. California had below average rainfall in the last seven or eight years, and downy mildews generally became uncommon except in the coastal fog belt of the San Francisco Bay area.

Because it has been suggested that the epidemic in Richmond, Indiana in August 1951 may have initiated from California-grown started-eye plants, the weather records during the period of production of these plants are of interest. Glasshouse roses are grown in Pleasanton, Livermore, San Fernando Valley, and Montebello, with a few sometimes in San Jose. Garden roses are produced at these places and also near Newman, Ontario, and Hemet. The rootstocks are grown from cuttings rooted in place in the field from October to February. These are budded in the following May to August, and the tops removed about two to eight weeks later. The "dormant-eye" plant is dug before the bud starts growth, and the "started-eye" type after the resulting shoot has branched. Plants remain in the California fields from about October of one year to October or January of the next.

The rainfall data for the principal California rose areas (or the nearest weather station to them) for the period during which the Indiana plants were being grown is given in Table 2. The

Table 2. Rainfall at eight locations in California during recent years.

Period	Rainfall in different localities						
	Pleasanton (Livermore data)	San Fernando	Montebello (Los Angeles data)	Newman	Ontario (Pomona data)	San Jose	Hemet
Annual average	14.00	16.54	14.76	10.06	19.34	13.93	13.48[b]
Average, Nov. through March	11.65	13.93	12.57	8.42	15.81	11.46	10.49[b]
Total, 1944	16.14	22.08	17.45	8.42[c]	18.97	16.46	13.30[b]
1945	14.25	16.72	12.78	10.72[c]	18.78	12.19	14.46[b]
1946	9.59	19.08	16.22	7.55[c]	20.23	9.01	12.68[b]
1947	15.57	4.94	4.13	4.61[c]	4.90	7.74	6.96[b]
1948	11.33	8.75	7.59	9.16	10.94	11.10	7.07
1949	10.33	9.27	10.63	7.66	13.81	9.83	7.95
1950	21.90	7.70	7.38	10.67	11.58	14.70	6.25
1951	17.14	15.24	14.33	10.27	16.72	12.21	11.19
Total							
Sept. 1950	.08	.45	.38	.23	.01	.11	.18
Oct.	1.84	.44	.24	.97	.23	1.61	.00
Nov.	5.95	1.06[a]	1.05	1.67	2.50	3.20	1.27
Dec.	4.95	Trace[a]	.03	3.01	.05	3.85	.00
Jan. 1951	2.23	3.38	2.80	1.39	2.57	1.87	1.42
Feb.	1.87	.83	1.48	1.92	1.19	1.80	.59
March	1.82	.72	.44	.26	1.02	.50	.56
April	.55	2.16	1.54	.76	1.87	.53	1.99
May through September	.41	.22	.39	.11	.35	.76	1.16
Oct.	1.04	.53	.59	.49	.49	.55	.57
Nov.	3.15	1.30	1.29	1.71	1.10	2.14	.62
Dec.	6.07	6.10	5.80	3.63	8.13	4.06	4.28

[a] Burbank data; San Fernando figures not available.
[b] San Jacinto data; Hemet figures not available.
[c] Los Banos data; Newman figures not available.

rainfall in all of these areas was so low during this period as to render unlikely an outbreak of downy mildew. The Pleasanton area had considerable rain in November (3.05 inches on November 19) and December (1.15 inches on December 7) 1950, but was extremely dry the rest of the time. The other areas were so dry during the period as to preclude downy mildew. The preceding years (Table 2) had been so dry as to make an accumulated high inoculum potential of downy mildew also improbable. Everything considered, it is most unlikely that California stock was the source of the Indiana outbreak. The fungus is known to have been present in the Central States for many years and its history shows many examples of reappearance at long intervals. There is little reason to believe that the Indiana outbreak is anything more than another such instance.

The 1952 Indiana Outbreak: The epidemic in August 1952 at Richmond, Indiana was more unusual in its environmental relationships. The disease appeared on California-grown plants that were benched on February 1, as well as on two and three-year-old plants. It occurred mainly in an old leaky ridge-and-furrow glasshouse section that had been kept humid through delayed opening of ventilators in the morning and early closure at night. The disease apparently was most severe in benches under the gutters and along the outside where drip was worst.

The weather records show below-average temperatures at Richmond for the months of June (-0.9°F.), July (-1.8°F.), and August (-0.8°F.). The average minimum temperatures for these respective months were 58.5°, 61.0°, and 59.1°F., and the average maxima were 77.7°, 81.9°, and 80.6°F. Table 3 shows that in June there were 19 days favorable to mildew (temperatures ranging between 40-80°F.), 8 days probably satisfactory (temperatures ranging between 50-85°F.), and only 3 days distinctly unfavorable (temperatures over 85°F.). In July the numbers of days were 12, 12, and 7, and in August, 17, 8, and 6, respectively. The rainfall in June was 5.78 inches (+1.77), in July 2.19 (-1.43), and in August 1.53 inches (-2.17). The records show no heavy rains or extended rainy periods during these three months. There was rainfall of .01 inch or more on 14 days in June, 10 in July, and 7 in August.

Table 3. Temperatures at Richmond, Indiana in summer of 1951.

Month, 1951	Number of days with maximum temperature of							
	80°F. or less and minimum of			81-85°F. and minimum of		over 85°F. and minimum of		
	40-49°F	50-59°F	60-70°F	50-59°F	60-70°F	50-59°F	60-70°F	over 70°F
June	2	11	6	0	8	2	1	
July	1	5	6	3	9	1	5	1
August	4	8	5	2	6	0	0	6

This low rainfall would not insure a "dry" glasshouse unless adequate humidity control was practiced. Heat was used in the range for 1-2 hours on 11 days, for 2.5-4 hours on 5 days, and for 5-6 hours on 2 days in the interval from July 1 to August 16. The partial records show night temperatures declining from 71-75°F. down to 62-69°F. in the infested houses. Calculations (2, 57) based on recorded day temperatures indicate that, assuming that the glasshouses had a daytime relative humidity of 70 percent or more and a night temperature of 65°F. or less, condensation could have occurred on 17, 27, and 24 nights during June, July, and August respectively. There were, in addition, 31 days with .01 inch or more of rain in these three months. Although no data are available on the relative humidity in the vicinity during the period, the weather summaries mention poor hay-curing weather in June, "numerous periods of humid weather" in July, and apparently a dry August.

The weather data, incomplete as they are, thus show no serious inconsistency with the outbreak of downy mildew on the basis of previously reported environmental relationships. It is more surprising that such temperatures occurred in Indiana in July and August than it is that the disease outbreak occurred. The situation may be similar to that reported for cucurbit downy mildew by Plakidas (36).

Varieties Affected

The varieties Red Delight and Pink Delight were the most seriously affected in the 1950-51 California outbreak. However, all varieties grown in those glasshouses with severe downy mildew showed some disease. In the Indiana outbreak Better Times was severely infected and Pink Bountiful somewhat less injured. Differences in varietal susceptibility have been reported by others (1, 12, 25, 27, 34, 44).

The fungus has been reported on the wild species, Rosa californica Cham. & Schlecht. (24), R. centifolia L. (33), R. canina L. (26), R. rubiginosa L. (26), and R. indica Lindl. (45).

Symptoms

The fungus occurs on leaves, stems, peduncles, calyces, and petals. Infected leaves develop purplish red to dark brown irregular spots, and the leaflets may become yellow; these symptoms may resemble a burn from fungicide or insecticide sprays. The whole symptom picture is sufficiently non-specific that the disease probably is more prevalent than its recognition would suggest. The purple color apparently was lacking on leaves in the Indiana outbreak. Infected leaves usually abscise, leaving a naked flower stem and rendering the blossom unsalable. The yellowing and rapid abscission of rose leaves infected by downy mildew suggests that ethylene is abundantly produced, as it is when they are invaded by Diplocarpon rosae or Cryptosporella umbrina (54). Under humid conditions, conidia and conidiophores appear copiously on the lower surface of the leaf; under less favorable conditions they may be so sparse as to be easily overlooked.

Purplish to black areas varying from small spots to areas an inch or more in length appear on the stems and peduncles. Similar spots and dead tips develop on the calyces. Infected twigs are sometimes killed (8, 22, 34, 35). The disease in California was not serious enough to kill any plants, nor were any petal symptoms produced. However, brown spots on petals of outdoor-grown roses have been reported in British Columbia (25), causing particular damage to young flowers.

Downy mildew on rose differs from the more common powdery mildew Sphaerotheca in that the former invades and usually kills host tissue, whereas the latter is external and kills tissue only when very severe and of long standing. Downy mildew causes spectacular leaf drop, which is much less severe with powdery mildew. The areas of leaves attacked by downy mildew are lighter in color, have more diffuse margins, and lack the radiate black fungus strands of those resulting from the black-spot fungus.

The Pathogen

The pathogen is nearly always reported as Peronospora sparsa Berk., and that species was involved in the 1950-51 California and 1951 Indiana outbreaks. The morphology of the fungus has been adequately described (7, 12, 16) and illustrated (5, 6, 7, 12, 18, 45). Another downy mildew, P. rosae gallicae Sav. & Rayss, has been reported in Roumania (46), but the description is very close to some of those given for P. sparsa.

Cuboni in 1888 (12) was the first to establish the pathogenicity of the fungus.

Oospores apparently were first found by Cuboni (12) in infected sepals and flower buds, but not in leaves and stems. Their occurrence seems to be quite variable, and Pickel (35) found that the fungus in Brazil lived over in stems as dormant mycelium without production of oospores. Cuboni (12) also reported survival of the fungus as dormant mycelium in the stem cortex. The California and Indiana material had abundant oospores that were about 18 microns in diameter in leaves and stems. Several workers (12, 26, 55, Eriksson in 42) have suggested that the fungus is distributed with cuttings and plants. Konopacka (26) has also suggested seed transmission.

Conidia may be produced over a long period of time from an infected leaf. Living detached leaves placed in a moist chamber on a north window ledge in Berkeley were still producing germinable spores one month after being picked. This characteristic, coupled with the enormous numbers of spores that may be produced on a single leaf under favorable conditions, provides a high inoculum potential. Cuboni (12) reported that the spores germinate in water in four hours, and that in a humid glass tube sporulation occurred on rose leaves within three days after inoculation. These conditions make possible the extremely rapid build-up to epidemic proportions that is so often evidenced by this fungus.

Control

The control procedures suggested for glasshouse roses by Berkeley (5) at the time of the first report of the fungus in 1862 are equally valid today: "..take care first that the house in which the plants are stored shall be well ventilated, that no syringing be used, and that no more water shall be given than is absolutely necessary, while care is taken that this shall go as little on the leaves as possible." Cuboni (12) again emphasized the importance of good aeration in control of the disease in 1888, and this has been reiterated many times since.

Control of the disease depends on maintenance of proper temperature and humidity. Humidity indicators, of which several types are available on the market, should be placed in various locations in the houses and read frequently. The principle should be followed of using heat to reduce humidity as well as simply to maintain proper temperatures. This is possible by opening the ridge ventilators to release the warm humid air (3). Special care should be taken at sunset to prevent a sudden slight temperature drop which may result in so great an increase in relative humidity as to result in condensation. The humidity should not be allowed to remain high for more than 3 to 7 hours, or infection may occur. Anything that can be done to decrease roof and gutter leaks, to improve air circulation and eliminate cold spots, and to reduce wet benches, walks, or floors during humid or cool periods is desirable.

Sanitation is a supplemental procedure to reduce the chance of carry-over of the fungus. Infected leaves should be raked up and burned, and infected stems should be pruned out and destroyed.

Many fungicidal materials have been applied at various times in efforts to control the disease. Among these are the following: sprays of sodium chloride (12), basic copper acetate (52), copper sulfate (12, 40, 52), copper oxychloride (30, 52), liver of sulfur (8), salicylic acid, soap, and alcohol (34, 52), lime (35), copper soda (9), copper soap (9), Bordeaux mixture (1, 9, 21, 22, 26, 32, 34, 35, 38, 44, 45, 49, 51, 52); dusts of sulfur (22, 55), copper acetate-sulfur (30); vaporization of sulfur (8, 21); treatment of soil with formaldehyde (14, 32, 34, 35, 52), copper sulfate (32), or Uspulun (32). Under glasshouse conditions entire dependence for control should not be placed on fungicides, but timely application of sprays may help reduce the build-up of disease. Sprays containing 2 lbs. of zinc ethylene bis-dithiocarbamate (Parzate or Dithane Z-78) per 100 gals. of water, plus an effective spreader-sticker, reduced the amount of downy mildew in severely infected glasshouse plantings in California in 1951.

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STEM NEMATODE DISEASE OF GREENHOUSE HYDRANGEAS

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In the spring of 1949[1], and to a lesser extent in 1951 and 1952, an unusual abnormality was found on greenhouse hydrangeas, Hydrangea opuloides Koch (H. macrophylla (Thunb.) DC), in Alabama. Since this disease has been the subject of only two brief reports, both in inaccessible European journals (1, 2), it would seem advisable to have a description and illustrations of the disease in a more readily available publication.

The disease, as found in March, 1949, was characterized by tip necrosis, scattered necrotic spots, and prominent stem hypertrophy (Figure 1, A through C). In late April, 1949, a few more plants were found which represented a less severe expression of the disease.

Several fungal saprophytes were isolated from diseased tissues. Since the symptoms were similar to stem nematode disease of phlox (3, 4, 5), the tissues were examined for nematodes. Active individuals of the genus Ditylenchus[2] were found.

In addition to the observations made in Europe (1, 2) the records of the Division of Nematology, Beltsville, Maryland, include only one probable observation of a Ditylenchus sp. on hydrangeas in the United States. These plants, observed in Salem, Oregon, in 1932, were growing next to nematode-infected phlox. These diseased hydrangeas in Alabama had never been near infected phlox. Careful investigation showed that the 1949 condition developed while the plants were being "forced" in the retailer's greenhouse. No evidence of the disease was found at the wholesale origin.

There is almost no indication that nematodes affect older stem tissues. (One prominent canker about one inch from the soil (Figure 1, D) could have been due to any of a number of agents.) Prolific development of normal branches from lateral buds (Figure 1, D, E), particularly the basal ones, indicate that lateral buds are not prominently infected by the nematodes. The invasion of the terminal bud must occur early in its development to be followed by a period of hibernation and incubation of the nematodes. The pathogens must have become active again during the "forced" growth of the hydrangeas when lateral buds had formed and become dormant as a result of terminal bud dominance. All of the reactions observed suggest centralization of the nematode's activity in succulent, non-dormant terminal meristematic tissue and stem parenchyma.

Symptoms

Two main types of reactions have been observed: (I) moderate infection involving stem elongation without hypertrophy (Figure 1, A through C), and (II) severe infection involving stem hypertrophy without elongation (Figure 1, D, E). Both types are accompanied by some necrosis and there are always indications that this disease develops partially as a result of practices associated with "forcing".

Severe Infection. In severe infection the most striking symptoms are necrosis of the terminal bud (Figure 1, B, C) and hypertrophy of new stem growth (Figure 1, A, B). As observed, the terminal bud was completely destroyed, leaving a blackened, indented stub at the stem tip (Figure 1, C). Enlargement in the slightly tapering fusiform galls was from one-fourth to one-half diameter.

Infected stem tissue is excessively succulent, but severely infected plants appear to be highly susceptible to wilting. Some such infected plants (Figure 1, A-left) could not be induced to recover from artificially induced wilting, while others recovered quite readily (Figure 1, A-right).

The epidermis of the hypertrophied stems changes from faintly chlorotic to distinctly yellowish, and streaked necrosis with prominent brown borders and yellow to white interiors develops. Eventually there are formed extensive and irregular blackened necrotic areas.

Petioles of older leaves also develop this bordered streak necrosis, but no hypertrophy.

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[1] The 1949 material was sent by the greenhouse owner to Mr. Henry P. Orr, Associate Professor of Ornamental Horticulture, the Alabama Polytechnic Institute.

[2] Identification by Dr. G. Steiner, Division of Nematology, U. S. Department of Agriculture whose cooperation in this and other respects is acknowledged.

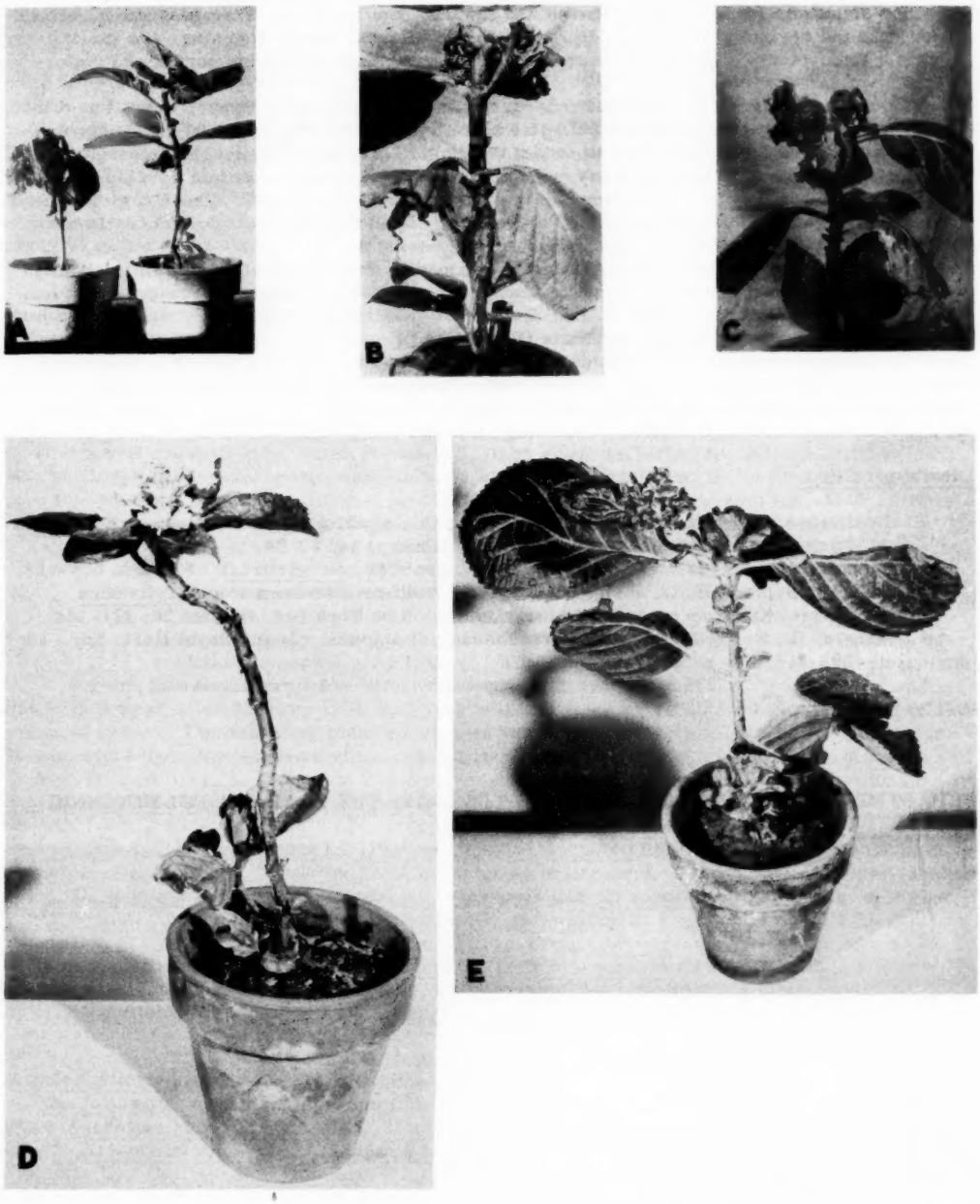

FIGURE 1. Stem nematode (Ditylenchus sp.) disease of greenhouse hydrangeas. A through C, severe infection; D and E, moderate infection. A -- Plants artificially wilted and then watered, lack of recovery (left) contrasted with recovery (right). B -- Fusiform gall, bud necrosis, and leaf-blade necrosis. C -- Closeup of bud and leaf-blade necrosis. D -- Stem elongation without hypertrophy, moderate stem necrosis, and leaf abscission. E -- Elongation of floral stalk, virescence of flowers, and proliferation of lateral buds.

Large necrotic areas also are found on blades of leaves of severely infected plants. Necrosis develops primarily from tip of the leaves and inward from margins. Irregular spots, either black from the beginning (Figure 1, B) or bleached yellowish then blackening, are the eventual results. Undoubtedly high temperatures and humidity during the forcing period are factors in this necrosis.

Moderate Infection. In moderate infection there is no detectable hypertrophy, but rather an appreciable elongation of either the entire new stem growth (Figure 1, D) or of only the floral axis (Figure 1, E). Considerable leaf abscission (Figure 1, D) accompanies moderate infection, but there is little leaf-blade necrosis. Bordered-streak necrosis develops on new stems and petioles (Figure 1, D), but rarely does it become blackened. There is pronounced stimulation of lateral bud development (Figure 1, E), even though apical dominance is not counteracted by death of the terminal bud.

Elongation from the terminal meristem is noticeably stimulated and apparently all meristematic tissue remains alive. Floral buds develop, but the resultant flowers are abnormal, small, and few in number (Figure 1, D). Petals are misshapen, and usually white, but sometimes much chlorophyll develops in petals (Figure 1, E).

Two years' experience with this disease indicates that control is obtained best through destruction of infected plants, soil sterilization, and other sanitation measures. Infected plants have been kept for two years under conditions of excellent sanitation. These plants have neither recovered nor died -- remaining alive but completely worthless.

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SOME EXPERIMENTAL HOSTS OF THE CHRYSANTHEMUM STUNT VIRUS

Philip Brierley

In the years immediately following the appearance of chrysanthemum stunt, about 1945 (2), an index plant, preferably one that could be produced from seed and that would express distinctive symptoms of this disease, was sought (1). The discovery of chrysanthemum varieties such as Mistletoe (3) and Blazing Gold (4), that express stunt clearly six to eight weeks after inoculation, simplified the problem of reselecting virus-free plants, and reduced the need for other test species. Nevertheless studies on host range of the virus were continued to determine whether other species of plants might be injured by stunt, or might serve as carriers of the stunt virus. Results of testing 76 species and varieties, largely members of the family Compositae, are reported here.

The chrysanthemum varieties Mary MacArthur and Mistletoe served as sources of stunt virus. Species and varieties of plants tested for susceptibility to stunt were for the most part grown from seed, but a few were produced from cuttings. Such seedlings or cuttings were inoculated when young and in vigorous growth; they were observed for about three months before return transfers to chrysanthemum test varieties were attempted. Generally 10 to 20 plants of each species were inoculated with stunt virus and like numbers of chrysanthemums were used in sub-inoculations; in a few instances numbers were smaller.

Groups of species were tested in parallel, each group including known susceptible plants such as florists' chrysanthemum varieties, to afford assurance that the conditions of test were favorable for infection. Manual inoculation with expressed sap, with the aid of carborundum powder, was the usual method; in a few instances graft transfers were made.

Chrysanthemum varieties used in tests to detect stunt on subinoculation from host range tests were Mistletoe and Mary MacArthur. The Mistletoe varieties -- Bronze, Golden, and Pink -- were virus-free when received in January 1951, and have never developed contamination with stunt or other viruses. The Mary MacArthur variety carried a low percentage of stunt when received in January 1949. Individual plants were numbered and the vegetative increases were carried as separate clonal lines. When any such line developed stunt contamination the entire line and all experiments involving this line was discarded. Stunt contamination ceased to appear after January 1951; and it is believed that our records have been freed of this source of error. The following plant species or varieties were found susceptible to chrysanthemum stunt from the evidence of successful recovery of stunt virus from them: Achillea millefolium L. (yarrow), A. ptarmica L., Ambrosia trifida L. (giant ragweed), Anthemis tinctoria L., Centaurea cyanus L., Chrysanthemum carinatum Schousb., C. cinerariaefolium (Trevar.) Vis., C. coccineum Willd. (received as Pyrethum roseum), C. coronarium L., C. corymbosum L., C. frutescens L. (Paris daisy), C. lacustre Brot. (Shasta daisy), C. leucanthemum L. (whiteweed), C. majus (Desf.) Aschers. (costmary), C. maximum Ramond (Shasta daisy), C. morifolium (Ramat.) Hemsl., C. myconis L., C. nivellei Braun-Blanq. & Maire, C. parthenium (L.) Pers. (white matricaria), C. parthenium f. flosculosum (DC.) Beck. (matricaria golden ball), C. praealtum Vent. (feverfew), Chrysanthemum sp. (received as Pyrethrum ptarmicaeflorum), C. viscosum Desf., Dahlia pinnata Cav., Echinacea purpurea (L.) Moench, Emilia sagittata (Vahl) DC., Heliopsis pitcheriana Hort., Liatris pycnostachya Michx., Sanvitalia procumbens Lam., Senecio cruentus DC. (cineraria), S. glastifolius L. f., S. mikanioides Otto (German ivy), Tanacetum boreale Fisch., T. camphoratum Less., T. vulgare L. (tansy), Tithonia rotundifolia (Mill.) Blake, Venidium fastuosum (Jacq.) Stapf, Verbesina encelioides (Cav.) B. & H., Zinnia elegans Jacq.[1]

The following were found insusceptible to stunt in parallel trials: Achillea filipendulina Lam., Ageratum houstonianum Mill., Ambrosia artemesiifolia L. (ragweed), Arctotis grandis Thunb., Artemisia vulgaris L., Boltonia latisquama Gray, Brickellia laciniata Gray, Calendula officinalis L., Callistephus chinensis (L.) Nees (China aster), Catananche caerulea L., Centaurea imperialis Hort., Cichorium intybus L. (chicory), Coreopsis douglasii (DC.) Hall, C. drummondii Torr. & Gray, C. lanceolata L., Cosmos bipinnatus Cav., Cynara cardunculus L. (cardoon), Doronicum cordatum (Wulf.) Sch. Bip., Echinops ritro L., Gaillardia picta Hort. Galinsoga parviflora Cav., Gazania hybrids, Gerbera jamesonii Hook., Helianthus annuus L., H. cucumerifolius Torr. & Gray, Helichrysum bracteatum Andr., Lactuca sativa L. (Slobolt

[1]The writer is indebted to J. L. Creech, Division of Plant Exploration and Introduction, for making available many chrysanthemum species and near relatives and to S. F. Blake, Division of Plant Exploration and Introduction, for classifying and naming the plants.

lettuce), Ligularia clivorum Maxim., Nicotiana glutinosa L., N. tabacum L., Phaseolus vulgaris L. (Topcrop bean), Rudbeckia serotina Nutt., Senecio elegans L., Stokesia laevis (Hill) Greene, Tagetes erecta L., Tragopogon porrifolius L. (salsify), Xeranthemum annuum L.[2]

Grafting served to introduce stunt virus into Chrysanthemum carinatum, C. coronarium, C. frutescens, C. parthenium f. flosculosum, Chrysanthemum sp. (received as Pyrethum ptarmicaeflorum), Tanaceteum camphoratum, and T. vulgare; grafting also recovered stunt virus from these species. Anthemis tinctoria and Chrysanthemum praealtum were sap-inoculated with the stunt virus; grafting reisolated the virus from these. Stunt was introduced into Chrysanthemum leucanthemum by grafting. Subinoculations by grafting failed to recover stunt virus from this species, but sap inoculations yielded stunt in 6 of 10 plants. Scions of C. leucanthemum had very short internodes; 6 survived for one month, but none as long as three months; thus failure to recover stunt virus by grafting seems due to poor unions.

In two tests, 45 plants of Chrysanthemum parthenium were not infected with stunt virus on sap inoculation and the virus was not recovered by this method. When graft-inoculated, 8 of 10 plants of this species developed rosetting symptoms and the virus was recovered in Mistletoe from 8 of 10 plants.

On 15 plants of Tanacetum vulgare sap inoculation also failed and the virus was not recovered. When 6 plants of this species were graft-inoculated with stunt scions of Mistletoe chrysanthemum survival of scions was poor but 2 of 6 scions lived for four months. On graft subinoculation 5 of 16 Tanacetum scions survived three months or longer on Mistletoe chrysanthemums; one of these scions produced typical stunt symptoms in the test stock. When Tanacetum camphoratum was graft-inoculated, 3 of 4 stunt scions lived four months or longer; after two months subinoculation by graft yielded 3 of 4 with stunt, all scions surviving to the end of the test (two months).

Only 7 of the 39 species and varieties listed as susceptible to chrysanthemum stunt developed recognizable symptoms. In addition of course the cultivated chrysanthemum is susceptible.

Plants of Chrysanthemum coccineum sap-inoculated with stunt virus were slightly paler and weaker than uninoculated controls. The virus was readily reisolated.

In Chrysanthemum morifolium, a primitive form received from Woodward, Oklahoma, inoculated plants were dwarfed and bore small pale leaves. Such symptoms are common in florists' and garden chrysanthemums. The virus was recovered easily.

Matricaria golden ball, when sap-inoculated in 1950, showed dwarfing, small pale leaves, small flowers, and shortened crowded inflorescences in all of 10 plants. The stunt virus was reisolated. When a second lot of 25 plants, grown from seed from a different seedsman, were sap-inoculated in 1951, symptom expression was poorly defined, and the number of plants infected could not be determined by inspection. On subinoculation by grafting to chrysanthemum 15 of 22 scions survived three months or longer but stunt was found in only 1 of 22 plants. In a third test with the 1951 stock of matricaria, symptoms were again poorly defined, and only 1 of 13 plants subinoculated by grafting proved to be infected with stunt. Matricaria golden ball is thus too variable or too lacking in sensitivity to be recommended as a test plant for detection of stunt virus. One-hundred thirty plants grown from seed of stunt-affected plants of the 1950 stock grew normally, without any evidence of seed transmission of the stunt virus. As stated above, the white matricaria proved graft susceptible to stunt, with symptoms similar to the golden ball variety, but was sap immune in our trials.

Chrysanthemum praealtum when sap-inoculated with stunt virus in 1949 developed rosetting in 2 of 6 plants which had small pale leaves, suggesting that direct diagnosis of stunt might be possible in this species. On graft subinoculation to chrysanthemum, however, 4 of the 6 plants were found infected. Thus stunt is not safely distinguished in populations of variable seedling plants. Nine seedlings from stunt-affected plants of this species proved stunt-free when graft indexed.

Senecio glastifolius, a coarse plant of little horticultural merit, was grown from seed and sap-inoculated with stunt virus in 1950. No symptoms developed after the first two months, and the virus was not reisolated in a test at that time. After eight months rosetting of shoot tips was evident; subinoculation to Mistletoe chrysanthemum at this time recovered stunt virus.

Senecio cruentus, the florists' cineraria, when first inoculated with stunt virus, in December 1949, developed distinctive local lesions in rubbed leaves of 17 of 20 plants after 30 days. Lesions were 1-3 mm. in diameter, round or angular, white or occasionally brown necrotic. Systemic effects first appeared after 35 days, becoming stronger after two to three months;

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[2]See footnote page 343.

affected plants were rosetted and had an upright bushy aspect, and leaves were small, pale, with the margins crinkled. The stunt virus was readily reisolated. One-hundred twelve plants grown for three months from seed from this series of plants showed no evidence of seed transmission of stunt virus.

Many attempts were made to utilize the local reaction in cineraria for detecting the stunt virus. Of 46 tests in which cineraria was inoculated from known stunt sources, 35 yielded some local lesions; 125 plants of 384 inoculated in these tests expressed lesions. Cinerarias from various sources, representing "Cremer's hybrids" and "Stellata" strains, reacted at times, but none did it consistently. Local lesions were produced December 1949 to May 1950, and again October 1950 to January 1951. In January 1951, 40 cinerarias were inoculated from Mistletoe chrysanthemums affected with stunt and 40 plants from cinerarias infected with stunt virus. None of the 80 plants showed local lesions but 32 of each set developed systemic effects. Thus cineraria is susceptible to stunt and consistent in expressing systemic symptoms, but local lesions are not sufficiently constant in expression to be useful in detecting the stunt virus.

Thus inoculation of 76 species and varieties of plants, largely in the family Compositae, revealed 39 susceptible and 37 insusceptible. Of susceptible kinds only seven developed recognizable symptoms -- five species or varieties of the genus Chrysanthemum and two species of Senecio. The others could serve as symptomless carriers of stunt virus if naturally infected. No test plant was found equal to the chrysanthemum varieties Mistletoe and Blazing Gold in speed and dependability of expression of stunt symptoms. No species of Chrysanthemum tested was found immune from stunt, but C. parthenium and the related Tanacetum vulgare showed resistance. Among the plants tested other than ornamental and wild ones, Chrysanthemum cinerariaefolium (source of pyrethrum insecticide) and C. majus (costmary, a flavoring herb) were found susceptible, whereas chicory, cardoon, lettuce, and salsify were not. No evidence of seed transmission of the stunt virus was found in 130 seedlings of Chrysanthemum parthenium f. flosculosum, 9 seedlings of C. praealtum, or 112 seedlings of Senecio cruentus grown from parents affected with stunt.

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APPARENT ABSENCE OF THE RING SPOT VIRUS IN THE PEACH VARIETY COLLECTION AT THE U. S. HORTICULTURAL STATION, FORT VALLEY, GEORGIA

Glenn KenKnight and Julian F. Jones

For use in determining whether the ring spot virus is present in peach varieties at Fort Valley, Georgia, Lovell peach seeds were planted at the U. S. Horticultural Field Laboratory in March 1952. In mid-September 1952, buds were taken from each of 142 orchard trees, mostly three to eight years old, representing 62 peach varieties growing on the laboratory grounds. Three Lovell seedlings were budded with 3 buds each from each source tree, and 1 seedling was left as a control. In addition, there were about 1,000 unbudded Lovell seedlings in neighboring rows. The seedlings were not pruned. They were examined March 2, 1953, when the leaves were beginning to unfold, and were reexamined at frequent intervals thereafter until March 23. Only about half the buds were alive March 2, but there appeared to be no relation between bud survival and source trees. A large majority of the test (budded) trees had at least one living bud.

There was no evidence whatever of delayed foliation on any of the budded trees. Careful examination revealed no ring patterns, chlorotic or necrotic spots, or shot-hole on the unfolding leaves of any of the test trees, and there was no evidence of rosetting of the foliage. Varieties such as Elberta (1870) and Hiley (1886), originating in the area and long grown here and in other parts of the Southeast, as well as such newer varieties as Cardinal, Dixigem, and Southland are maintained in the variety orchard sampled. This test indicates that ring spot is not present in the indexed trees.

The list of varieties indexed follows:

Fruit Varieties:
- Autumn
- Cardinal
- Cherryred
- Dixigem
- Duckers Sport
- Early East
- Early Fairs Beauty
- Early Hiley
- Early Triogem
- Erly-Red-Fre
- Elberta
- Fairhaven
- Fowler
- Goldeneast
- Goldenglobe
- Halegold
- Halehaven
- Hawaiian
- Hiley
- Jerseyland
- J. H. Hale
- July Elberta
- Loring
- Mayflower
- Missouri
- Newday
- Osage
- Poppy
- Prairie Dawn
- Prairie Daybreak
- Prairie Rose
- Prairie Sunrise
- Redcrest
- Redhaven
- Redskin
- Rio Oso Gem
- Shinn's Delicious
- Shippers Late Red
- Southland
- Stacey Elberta
- Stack
- Starking Delicious
- Stoner
- Sullivan Elberta
- Summerrose
- Sunhigh
- Triogem
- Tulip
- Vanguard
- Walker Elberta
- White June
- Wildrose
- Wilson
- World's Earliest

Ornamental Flowering Peaches:
- Dark Pink
- Peppermint
- Red
- White

Varieties Producing Seed for Rootstocks:
- Bound Brook Green Leaf
- Rutgers Red Leaf
- Tennessee Red Leaf
- Yunnan

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, UNITED STATES DEPARTMENT OF AGRICULTURE, FORT VALLEY, GEORGIA

POTATO CALICO IN MAINE

Donald Folsom

So far as is known, the only authentic instance, previous to the one to be described in this report, of occurrence of potato calico in Maine is the one in which calico was introduced by E. S. Schultz from California in 1930 for experimental tests in northeastern Maine (6, p. 284).

Folsom (2, p. 217), during the roguing of hundreds of thousands of hills, observed about one hill in every 40,000 to be a sport with respect to color, form, and hairiness of foliage. Such sports reverted to normal form wholly or in part when perpetuated through one or more generations. The effect of one sport on yield rate was found to be a reduction by 18 percent in a comparison of the sport part of a tuber line with a part of the same tuber line that had not sported and with a part that had reverted from sport to normal.

A yellow-mottled condition, resembling some sports, was found in 1949 in a field in southwestern Maine in scattered hills of the varieties Green Mountain, Katahdin, and X1276-185 (a seedling variety grown to some extent commercially in Maine). In 1950 the progeny of the Green Mountain hill showed no mottling, while the mottling reappeared in the Katahdin and seedling progeny and also appeared in a Chippewa hill growing 3 feet distant from the mottled Katahdin stock. In 1951 the mottling reappeared in the Katahdin, seedling, and Chippewa stocks. The study was continued in the seedling in 1951-52 in the greenhouse, where plants showed very little mottling, although the same stock showed conspicuous mottling in the field in 1952. The tubers produced by the 1951-52 greenhouse plants were planted in the greenhouse in 1952-53 and the plants grown from them were conspicuously mottled. These plants were used for sap and carborundum leaf inoculations of young Green Mountain and seedling plants. In four weeks, half of the inoculated Green Mountain plants showed some mottling and all of the inoculated seedling plants showed conspicuous mottling (Fig. 1). Uninoculated plants of the same tuber units were not mottled except for one which showed mottling in the youngest leaves a month after the mottling appeared in the inoculated plants. Aphids had been allowed to accumulate to some extent, before being controlled by parathion, when the plants were young. The mottle symptoms were identical with those attributed by Porter to calico (6, Fig. 6 and Plate 1). Porter (5) transmitted calico to plants of the Green Mountain variety and a dozen other varieties. Black and Price (1) considered potato calico virus to be closely related to alfalfa mosaic virus, infection by the former being found to protect against infection by the latter. Oswald (4) considered calico virus and a tuber-necrosis virus to be strains of alfalfa-mosaic virus, the calico protecting against infection by the tuber-necrosis.

Parallel inoculations in the Maine greenhouse with the tuber-necrosis virus received from Oswald produced symptoms typical of the tuber-necrosis virosis on potato and very distinct from calico symptoms, except that occasionally there was some yellowing (Fig. 2 and 3) of leaflets closely resembling the most severe yellowing of leaflets of calico plants in the same tuber units. This yellowing, stated by Oswald to be absent in tuber-necrosis virosis, had been observed also in the winter of 1951-52 following tuber-necrosis greenhouse inoculations in the absence of calico inoculations.

Oswald found both viruses to spread to potatoes from alfalfa and Ladino clover fields. The original source of the Maine calico in 1949 might have been Ladino clover on the same farm. None was near the potatoes and the clover was not examined.

The tuber-necrosis stock from Oswald in California was used in the Maine greenhouse in the winters of 1950-51, 1951-52, and 1952-53 to see how similar it was to unmottled curly-dwarf (3), which it resembled. Tuber-necrosis virus had no spindle-tuber effect on tubers, while unmottled curly-dwarf has an extreme spindle-tuber effect. Oswald (letter of April 28, 1952), in his work with unmottled curly-dwarf virus from E. S. Schultz, found bean and White Rose potato to be unreceptive to this virus, showing it to be different from alfalfa-mosaic strains.

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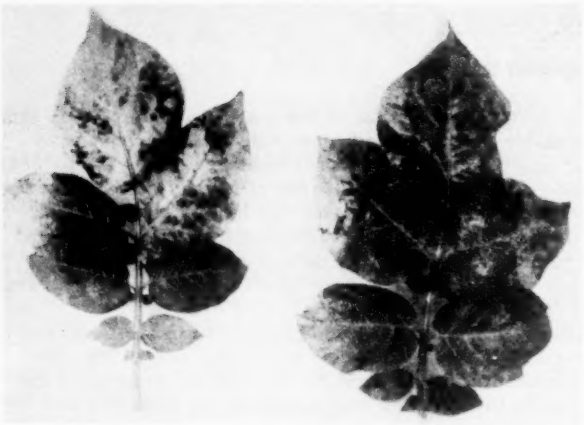

FIGURE 1. (left) Inoculative symptoms of calico mosaic on potato seedling variety X1276-185. (Copy of Kodachrome made by projecting on SS film with an enlarger.)

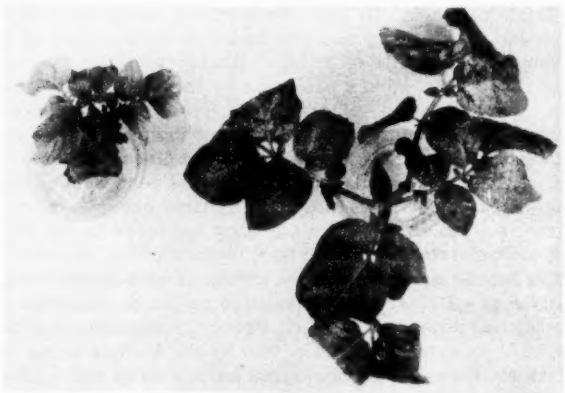

FIGURE 2. (right) Inoculative symptoms of calico mosaic (in the larger shoot) and the tuber-necrosis strain of alfalfa mosaic on potato seedling variety X1276-185. (Copy of Kodachrome made by projecting on SS film with an enlarger.)

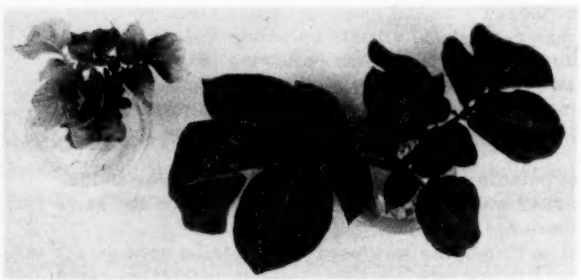

FIGURE 3. (left) Inoculative symptoms of the tuber-necrosis strain of alfalfa mosaic vs. healthy shoot tip of potato seedling variety X1276-185. (Copy of Kodachrome made by projecting on SS film with an enlarger.)

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MAINE AGRICULTURAL EXPERIMENT STATION, ORONO

REACTION OF AGROTRICUM HYBRIDS TO THE VIRUS OF YELLOW STREAK MOSAIC OF WHEAT[1]

Hurley Fellows[2] and John W. Schmidt[3]

Research to date has revealed that none of our common varieties of wheat has enough resistance to streak mosaic to be economically important in that respect. Accordingly, the search has been extended to crosses between commercial wheat varieties and related grass species[4]. The experiments reported in the paper were with crosses between Agropyron species and wheat. The work was done in the Botany greenhouse at Manhattan, Kansas, during the winter of 1951-1952.

MATERIALS AND METHODS

All plants were grown in 4-inch pots with four plants per pot. Two consecutive tests were run, involving duplicate and triplicate pots of each Agrotricum strain. Accordingly, 8 plants of each strain were tried in the first test and 12 in the second. In the first test 51 strains were tested, and in the second, 54. The second test included 23 strains that appeared especially favorable in the first.

At about the four-leaf stage the plants were inoculated by the wiping method with the virus of the yellow streak mosaic of wheat, Marmor virgatum McK. Five observations were made at varying intervals after symptoms started to show. At the end of the fifth observation all plants showing symptoms were destroyed and the remaining symptom-free plants reinoculated to detect chance escapes. The basis of selection was on the presence or absence of mosaic mottling. The final readings were made 20 days after the last inoculation. The checks used were Marquillo-Oro x Pawnee (C.I. 12851), [5] a very susceptible variety, and Triumph (C.I. 12132) a variety found to have some resistance. Agropyron elongatum (Host) Beauv. and A. intermedium (Host) Beauv. also were inoculated.

There were four different wheat-by-Agropyron crosses tested with several strains of each. In the cross (Chinese x A. elongatum) x Pawnee (F_4) the Chinese x A. elongatum cross was secured from L. H. J. Shebeski of the University of Saskatchewan, Saskatoon, Canada. The cross of this to Pawnee was made at Manhattan, Kansas, by John W. Schmidt in 1948. The crosses wheat by A. elongatum and wheat by Agropyron were made by W. J. Sando. The wheat by A. trichophorum (Link) Richt. hybrids were obtained from C. A. Suneson, University of California, Davis, California.

RESULTS

According to the data presented in Tables 1, 2, and 3, the hybrids show a wide diversity of reaction to the virus of yellow streak mosaic as based on the presence or absence of mottling ranging from complete resistance through segregation to complete susceptibility. Four of the resistant hybrids were reported by McKinney and Sando[6]. According to them, three of the four were resistant and one segregating but mostly resistant. The four were all resistant in the tests at Manhattan. Random samples taken from symptomless plants showed the virus to be present so there was no complete immunity. However, these hybrids appear to be a probable or favorable source of resistance to mosaic. The tables indicate further that the

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[1]These investigations were conducted cooperatively between the Division of Cereal Crops and Diseases, Bureau of Plant Industry, Soils and Agricultural Engineering, United States Department of Agriculture, and the Kansas Agricultural Experiment Station, Agronomy Department, Kansas State College. Contribution No. 543, serial No. 427 Department of Botany and Plant Pathology, and Contribution No. 487 of the Agronomy Department, Kansas State College.

[2]Pathologist, Division Cereal Crops and Diseases, United States Department of Agriculture.

[3]Assistant Agronomist, Kansas Agricultural Experiment Station, Manhattan, Kansas.

[4]McKinney, H.H., and Sando, W.J. 1951. Susceptibility and resistance to the wheat streak-mosaic virus in the genera Triticum, Agropyron, and Secale and certain hybrids. U.S. Dept. Agr., Bur. Plant Indus., Soils and Agr. Engin. Plant Dis. Reptr. 35: 476-479.

[5]C.I. refers to accession number of the Division of Cereal Crops and Diseases.

[6]Loc. cit.

Table 1. The reaction of 105 Agrotricum strains to manual inoculation with the virus of yellow streak mosaic of wheat.

Cross	Total strains tested	Reaction of cross strains: Number			Percent		
		Res[a]	Seg	Sus	Res	Seg	Sus
(Chinese[2] x A. elongatum) x Pawnee	14	0	12	2	00.0	85.7	14.2
Wheat x A. elongatum	81	41	28	12	50.6	34.6	14.9
Wheat[3] x Agropyron	8	3	3	2	37.5	37.5	25.0
Wheat[3] x A. trichophorum	2	0	1	1	00.0	50.0	50.0

[a] Res. Resistant meaning no plants showed symptoms.
Seg. Segregating. Some plants showed symptoms, others not.
Sus. Susceptible. All plants showed symptoms.

Table 2. The reaction of plant types found among 105 Agrotricum strains to manual inoculation with the virus of yellow streak mosaic of wheat.

Plant Type	Total strains tested	Reaction of cross strains: Number			Percent		
		Res	Seg	Sus	Res	Seg	Sus
Grasslike	11	8	3	0	72.7	27.2	00.0
Intermediate	74	36	26	12	48.9	35.1	16.0
Wheatlike	20	1	15	4	5.0	75.0	20.0

Table 3. The similarity of response according to plant type of 23 Agrotricum strains which were tested twice for their reaction to the virus causing yellow streak mosaic of wheat.

Plant Type	Number retested	Number of strains and reaction: Test one			Test two		
		Res	Seg	Sus	Res	Seg	Sus
Grasslike	3	3	0	0	3	0	0
Intermediate	18	13	5	0	11	5	2
Wheatlike	2	0	1	1	0	1	1

more wheat-like the hybrid, the less resistance is retained. However, the wheat-like types retain a high percentage of variability, suggesting that it should be possible to select resistant wheat-like strains. Details of the data taken but not given in the tables show that within segregating strains, individual plant reactions vary from high level resistance to nearly complete susceptibility. The degree or severity of mottling also varied greatly as did the time required for symptoms to appear after inoculation.

Neither the highly susceptible nor the somewhat resistant wheat checks used showed complete susceptibility as determined by the presence or absence of mosaic symptoms. Among the inoculated Triumph plants, 26.3 percent were free of symptoms as were 6.2 percent of C. I. 12851. It has been observed in other experiments that it is seldom that all inoculated Triumph plants show symptoms. Often symptoms on certain Triumph plants appear tardily and many plants have only mild symptoms. Both Agropyron species used as controls were free of symptoms.

DIVISION OF CEREAL CROPS AND DISEASES, U. S. BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AND KANSAS STATE COLLEGE

USE OF CARBON DIOXIDE AS AN ANESTHETIC IN INSECT VECTOR WORK

Karl Maramorosch

It has long been known that insects are rapidly anesthetized by carbon dioxide and that recovery occurs quickly. Since 1937 l'Heritier used CO_2 routinely in France for genetic studies of Drosophila (3). Williams (8) described a method of prolonged anesthesia of insects with carbon dioxide. Ribbauds (6), studying changes in the behavier of honey bees induced by various anesthetics, concluded that carbon dioxide and nitrogen stop the respiration of insects, whereas ether and chloroform act on the nervous system. This note describes the use of carbon dioxide anesthesia for routine handling of leafhoppers and for mechanical virus transmission to leafhopper vectors. In a number of respects this anesthetic appears to be superior to those previously used.

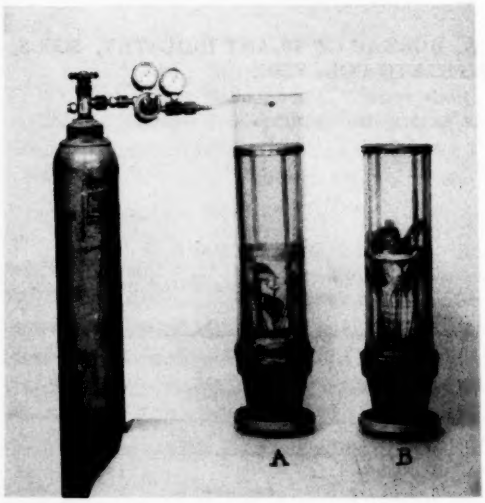

FIGURE 1. Method used to anesthetize caged leafhoppers. A. Insect cage covered with plastic hood, connected to a cylinder containing CO_2. B. Cage without hood; galvanized iron frame is covered with a Lumite screen. (Photograph by J. A. Carlile.)

Ethyl ether anesthesia usually results in a small percentage of loss due to the death of insects, whereas no loss has resulted from treatment with carbon dioxide. Furthermore, ether and chloroform have an unpleasant and nauseating odor. Although no deleterious effect of ether on plant viruses has been reported, certain unstable insect-borne plant viruses may possibly be affected by it, as are some animal viruses. On the whole, the animal viruses which were found ether-resistant are "tough" viruses, which can be left at room temperature without fear that they will quickly lose activity; the ether-sensitive ones are, in general, more delicate and some are ether-sensitive to a high degree, especially arthropod-transmitted viruses of yellow fever (1) and St. Louis, Eastern and Western equine encephalomyelitis (7).

Prior to 1953, mechanical transmission of plant viruses to leafhopper-vectors was usually done in a coldroom at 0°C. (2, 4, 5). With the use of CO_2 the same operation can be made at temperatures ranging from 4° C. to 15° C. or even at room temperature. The procedure is very simple. Insects are placed in glass tubes and CO_2 introduced from a cylinder. Within a few seconds the insects are immobilized and can be removed from the tubes and inoculated. Temperatures at or below 15° C. were found more desirable than room temperature as the insects often recover too quickly at the higher temperature. With the very labile aster-yellows virus, the percentage of "takes" at 15° C. and at 0° C. did not differ when the inoculum was kept in ice water before use. The application of CO_2 anesthesia has eliminated the necessity of working in a coldroom.

In collecting large numbers of insects from caged plants, it was desirable to use an anesthetic harmless to both plants and insects. Carbon dioxide fulfills these conditions. It is also superior to nitrogen, being heavier and thus easily filling the bottom of a container or cage with insects. In addition, it is economical to use. Anesthesia with carbon dioxide was found very helpful in routine handling of large numbers of stock insects, especially of certain very

lively species of leafhoppers like Macrosteles fascifrons, the aster leafhopper, or Circulifer tenellus, the beet leafhopper. For this purpose a celluloid hood is placed over caged plants with insects. As shown in Fig. 1 A, the gas is introduced into the enclosure through a rubber tubing. After a few seconds the hood and cage can be removed and as many as 200 to 500 immobilized adult leafhoppers can be collected within one minute. After the removal of insects it is advisable to recage the plant to prevent the possible escape of individuals left on the foliage or on the soil. Small cages can be covered more simply with a paper bag than the celluloid hood. The cage represented in Fig. 1 B, made of a galvanized iron frame covered with plastic screen, was found very convenient for anesthetizing insects and also for rearing certain species of leafhoppers that require low humidity.

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OBSERVATIONS AND EXPERIMENTS ON SNOW MOLD OF WINTER WHEAT IN WASHINGTON STATE[1]

C. S. Holton[2]

Snow mold[3] has long been a hazard to winter wheat production in localized areas of the Pacific Northwest (24) and adjacent territory (8). The reports of Heald (2), Hungerford (4), Remsberg and Hungerford (6), and Young (8) imply a greater prevalence of this disease in areas of relatively high altitude where snow falls early on unfrozen soil and remains for long periods (three to five months). Since about 1940, however, both formal (1) and informal reports on this disease indicate a wider regional prevalence and also an apparently less consistent association with snow cover. Similar, more recent, reports (37) of winter damage resembling snow mold suggest a relentlessly increasing prevalence of this disease, with a correspondingly increased concern over the damage it causes. The current situation emphasizes the need for additional published information on the nature of the snow mold disease in relation to the possibilities for its control. This is a report of observations and studies on snow mold of winter wheat in Washington in the period 1945-48.

Kinds of Snow Mold in Washington

Two types of snow mold, caused by three species of fungi, were prevalent in this area during the period of these observations. Although all three species were found commingling throughout most of the area, there were differences in prevalence and severity that appeared to be related to snow cover. In areas of heavy snow cover of long duration, the most prevalent species was Typhula idahoensis Remsberg. It causes snow scald (5) or the so-called speckled snow mold. In transitional areas between heavy and light snow cover, Fusarium nivale (Fr.) Ces., which causes pink snow mold, was predominant. In areas with light snow cover of relatively short duration, Typhula itoana Imai predominated. Blodgett (1) reported similar observations for the first two species in southern Idaho. Except for the apparent relationship to snow cover, the specific ecological factors involved in the distribution of the above species in Washington were not determined. In the period covered by these observations, T. idahoensis caused the most damage and the experiments reported here relate almost entirely to this species.

Experiments in 1944-45

Plots were established in heavily infested soil on two farms near Mansfield, Washington, to determine whether snow mold can be controlled or reduced in severity by certain cultural practices or by use of resistant varieties. Fifteen varieties and hybrid selections of winter wheat were sown in duplicate rows 16 feet long. Comparable plots were seeded by the deep-furrow and common drill methods on clean fallow and on stubble mulch. Seedlings were made on two dates, September 9 and September 30, 1944. Good stands were obtained and winter conditions were ideal for development of snow mold. Readings on disease severity were made on March 22 and June 22, 1945.

Snow mold (Typhula idahoensis) developed severely throughout the plots. The results exhibited in March indicated that none of the cultural practices had any appreciable influence on the disease severity. The disease developed equally well on stubble mulch and clean fallow, from both dates of seeding, and from both methods of seeding. All varieties appeared to be equally severely damaged.

Observations the following June revealed that apparent damage in early spring is not necessarily indicative of final damage to the crop. Although damage appeared to be severe enough in March to require reseeding of these plots, there was sufficient recovery by June to insure a good crop. Furthermore, it appeared that certain varieties had greater recoverability than

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[1]Cooperative investigations of the United States Department of Agriculture, Bureau of Plant Industry, Soils, and Agricultural Engineering, Division of Cereal Crops and Diseases, and the Washington Agricultural Experiment Stations. Project No. 796.

[2]Pathologist, Division of Cereal Crops and Diseases, Pullman, Washington. The writer acknowledges with appreciation the assistance of Dr. J. P. Meiners in establishing some of the fungicide plots.

[3]As used here, snow mold is a general term applied to a disease of grains and grasses usually, but not always, associated with snow, which has been referred to variously as snow mold, snow scald, and snow rot.

others. This suggested a possible basis for varietal recommendations to reduce snow mold damage.

Observations and Experiments in 1945-46

Variety Tests: Varietal tests were sown on three farms to explore the possibility of reducing losses from snow mold by growing varieties with resistance or with high recoverability. Sixteen varieties and hybrid selections were sown in 3-row plots, 20 feet long, and replicated twice. The seeding was done in mid-September and a good stand was obtained. Winter conditions were ideal for development of snow mold.

Observations in early April, 1946 showed almost a complete kill of all varieties in all nurseries. The Wasatch variety survived in trace amounts in two nurseries but not in the third one. Plans to observe the varieties for differential recoverability in late spring were necessarily abandoned. These results appeared to eliminate the possibility of reducing snow mold damage in this area by growing resistant or tolerant varieties.

Fungicide Tests: Tests were made to determine the effectiveness of certain chemical dusts, applied to the foliage just prior to snowfall, in controlling snow mold. Four copper dusts, one sulfur dust, Ortho K Dust No. 25, Fermate, Phygon, and Spergon were used. These dusts were applied with a small wheelbarrow-type duster which forced the material from a hopper through six flexible tubes each of which was aimed directly over a drill row of wheat. The center row, over which the wheel passed, received no treatment and thus served as a check, as also did the area surrounding the plots. Each dust was applied at the maximum capacity of the machine, but the rate varied according to the nature of the dust. All rates of application were recognized as economically prohibitive; however, the main purpose of the tests was to determine whether any of the materials would be effective under conditions favoring development of snow mold.

Only Spergon and Phygon exerted any control action against the disease. This was evidenced by a stand of about 50 percent of disease-free plants in these plots as contrasted to only a trace of good plants in the check plots and in plots of other treatments. These results were promising enough to justify further tests with these and other compounds.

Observations on Recovery of Diseased Plants in Commercial Fields: Early sown wheat appeared to withstand snow mold better than did that sown late. It was evident that large plants recovered more readily and completely than did small ones. The smaller plants from later seedings, where tillering was at a minimum, usually were completely destroyed when attacked by the disease. In contrast, only the top growth of the older, vigorously tillering plants was destroyed; and these plants invariably recovered and produced grain. This differential reaction of individual plants from early and late seedings was exhibited throughout the infested area. Several early sown fields that appeared to need reseeding when checked in April were checked again in July. By then the diseased plants had recovered sufficiently to produce a good crop. Two fields of Turkey wheat seeded in July showed almost a complete kill of top growth the following April but recovered sufficiently to produce a 20-bushel crop. In contrast, the diseased plants in fields seeded six to ten weeks later had poor recovery, as indicated by spotty stands, weedy conditions, and low yields. While this might indicate that snow mold can be combatted by early fall seeding, spring wheat seeded on the same land probably would yield up to 10 bushels more grain an acre.

The degree of recovery of apparently severely damaged plants was observed. Individual diseased plants were selected in April and the extent of damage was estimated in percentage of top growth destroyed. Healthy plants growing under comparable conditions also were marked for later observation. By July all plants that showed 75 to 90 percent damaged in April had recovered sufficiently to produce mature plants of apparently only slightly less vigor than those marked healthy.

In commercial fields, one 16-foot strip with an estimated 15 percent stand in April had an estimated 75 percent stand in July. A circular area 10 feet in diameter with less than a 10 percent stand in April had a 75 percent stand in July. In both cases, however, the plants were later and less vigorous than healthy ones and the plots were weedy, indicating severe damaging effects of the disease despite apparent recovery. The difference in growth and vigor of diseased and healthy plants of the same age grown under the same conditions is shown in Figure 1.

FIGURE 1. Turkey wheat plants of the same age showing effects of snow mold on growth and vigor. Left, diseased plants; right, healthy plants; as they appeared in May.

FIGURE 2. Center background -- effectiveness of Phygon in controlling snow mold of winter wheat. Center foreground -- untreated check.

FIGURE 3. Protective value of Spergon against snow mold of winter wheat. Left from Spergon-treated plots; right from untreated plot. May, 1948.

Aug. 5 Aug. 19 Sept. 8 Sept. 22

FIGURE 4. Snow-mold affected plants from four seeding dates showing greater recoverability of the larger plants. Note dead leaves from fall growth at base.

Observations and Experiments in 1947-48

No snow mold developed in 1946-47, owing to generally mild weather and lack of snow. Consequently no data were obtained from the experimental plots. The tests with fungicides and seeding dates were repeated in 1947-48, when snow mold was widely prevalent and severe throughout the area.

Fungicide Tests: Spergon and Phygon were applied to two plots each covering an area 40 X 80 feet in a commercial field seeded September 8, 1947. The dusts were applied with the equipment already described.

Table 1. Effect of fall-applied fungicides on snow mold control. 1947-48 season.

Treatment	Percent stand
Spergon (Foliage cover only)	89
Check (No treatment)	3
Spergon (Foliage cover plus ground cover)	96
Check (No treatment)	2
Phygon (Foliage cover only)	89
Check (No treatment)	1
Phygon (Foliage cover plus ground cover)	91
Check (No treatment)	5

The rate of application was the maximum delivery capacity of the duster, which was about 80 pounds per acre for these dusts. Both dusts were applied as a foliage cover and as a foliage cover plus ground cover between rows. The dusts were applied on October 17, 1947. Notes taken in March 1948 indicated good control of the snow mold in these plots by both Spergon and Phygon. (Figures 2 and 3). The percentages of diseased plants in treated and untreated plots in April are presented in Table 1. The results confirmed those of the 1945-46 test in which both Spergon and Phygon effectively controlled snow mold. However, these materials are now too expensive for practical use for this purpose. Sprague (7) has since found that Ceresan M provides economical control of this disease.

Effect of Plant Size and Date of Seeding on Snow Mold Injury: Plots of one acre each established on the Warren Gallaher ranch in Douglas County near Mansfield, Washington, were sown on five dates at two-week intervals between August 5 and October 6, using a deep furrow drill. The extent of snow mold damage, as indicated by destruction of top growth on March 29 and the degree of recovery on June 26 are summarized in Table 2.

It is evident from these observations that the larger plants from early seedings are better able to withstand the onslaught of snow mold and to recover and produce a fair crop than are the smaller plants of later seedings. While the late-sown crop tends to escape snow mold infection, most of the infected plants are killed. Furthermore plants from late seeding that escape are too small and weak to compete with weeds and produce a crop. The relative vigor of plants from four dates of seeding and the recovery from snow mold is illustrated in Figure 4.

Miscellaneous Tests: Three tests were conducted to determine whether snow mold damage could be reduced by measures taken in early spring before the snow melts. Ammonium nitrate fertilizer was applied on the snow at different rates to determine whether it would stimulate the diseased plants and thus contribute to their recovery and vigor. Although there was no evidence of any beneficial effect in this test, later tests of similar treatments indicated that they aided recovery of diseased plants (3).

In another test, the snow was shovelled off to determine whether early disappearance of snow would reduce the disease severity. No beneficial effects were evident. The plants were damaged and stands reduced as much or more in these plots as in those where snow remained until it melted.

Lamp black was applied to the snow on several plots to hasten melting and raise soil temperatures. In these plots there was enough evidence of early enhanced vigor of the plants and apparent beneficial effects to suggest a need for further tests.

Table 2. Snow mold damage on March 29 and recovery on June 26, 1948, of winter wheat seeded on five dates in the fall of 1947.

Date seeded 1947	Indicated damage and recovery
Aug. 5	All foliage destroyed but no plants killed. Recovery of all plants by sending out new shoots from the crown.
Aug. 19	Severe damage with plants apparently dead. Partial recovery but with reduced stand.
Sept. 8	Severe damage, indicating complete kill of almost all plants. Partial recovery with a thin stand of plants lacking in vigor and unable to compete with weeds.
Sept. 22	Severe damage with indications of complete killing. Recovery to a poor stand of weak plants unable to compete with weeds.
Oct. 6	Plants very small with no tillers and no appreciable snow mold damage, most of the plants apparently having escaped infection. Infected plants dead and those surviving by escaping infection were too weak and retarded to compete successfully with weeds and produce a crop.

Summary

1. The fungi found associated with snow mold of winter wheat in Washington were Typhula idahoensis, T. itoana, and Fusarium nivale.

2. Snow mold developed equally well under several common cultural practices of the infested area, including stubble mulch, clean fallow, burned stubble, deep furrow drill, and common drill.

3. There was no difference in varietal reaction to snow mold. All varieties tested were highly susceptible under all conditions encountered in the tests.

4. Apparent damage from snow mold in early spring usually is more severe than the real damage as exhibited in late spring and summer, owing to recovery of diseased plants.

5. Growth and vigor of recovered diseased plants is in direct relation to plant size. Large plants from early seedings are more tolerant of snow mold damage than are small plants from late seedings.

6. Snow mold of winter wheat can be controlled by heavy foliar application of Spergon and Phygon prior to snow cover in the fall. Economical rates of application of these materials were not determined.

7. Slight beneficial effects, as exhibited by recovery of diseased plants, were produced by early spring application of ammonium nitrate and lamp black. Early mechanical removal of snow did not reduce the disease.

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DIVISION OF CEREAL CROPS AND DISEASES, UNITED STATES BUREAU OF PLANT INDUSTRY, SOILS AND AGRICULTURAL ENGINEERING AND WASHINGTON AGRICULTURAL EXPERIMENT STATIONS

FUNGICIDAL CONTROL OF SNOW MOLD OF WINTER WHEAT IN WASHINGTON

Roderick Sprague

Such materials as Phygon, Spergon, and Puraturf used at heavy and costly rates gave control of snow mold (Typhula spp. and Fusarium nivale) in eastern Washington as early as 1948 (1). After it was found that basidiospores were discharged, usually in mid-November (2, 4), and that these were important in causing mold in the wheat (3), an effort was made to obtain an effective fungicide to protect the wheat during the time of the spore shower in November. Ceresan M gave promise in the spring of 1951 in a field with very light infection. In 1951-52, 4 lbs. 11 oz. per acre of Ceresan M in a lightly dampened pumicite dust gave striking control in 1/20-acre strips.

In the third year's tests, in 1952-53 near Waterville, Washington, control observed in the plots on March 14 and rechecked on March 19 was again good with Ceresan M at 5 lbs. per acre. However, control was less dependable in the field in larger plots treated by plane. The weather during the winter wheat season of 1952-53 was considerably different from average. The summer and fall of 1952 had been very dry and basidiospores were not discharged until almost December. The winter was mild and snow melted in early March over most of the area. The soil surface, instead of drying out, remained somewhat damp because scattered wet snows continued to fall. The plots treated by plane were not uniformly covered by the treatment. Portions in swales or behind hillocks tended to show some infection in early March. By March 19 vegetative reinfection had spread appreciably in some areas and it was uncertain as to just how effective the treatment had been. This part had been treated with the usual 5 lbs. of Ceresan M in 40 lbs. of NuGreen (urea) fertilizer per acre. This combination, from the viewpoint of the operator, has proved to be ideal. The NuGreen was pelleted and the relatively small amount of Ceresan M was carried without evident dust. The combination had been suggested by Phil Wainscott and his father Paris Wainscott who had the material applied on a small commercial scale on their holdings near Waterville. In company with them and County Agricultural Agent Jack Ramsey, and later, Extension Plant Pathologist Marion Harris, conclusions as to the value of the test are being made. By April 8 the treated plot does not appear as healthy as it did when it came out from under the snow several weeks ago. However, the crowns of Ceresan-treated plants are appreciably healthier than those given fertilizer alone. Until yield data can be obtained, the commercial value of our tests will not be decided. If reseeding to spring wheat, as is often necessary in this area, can be avoided the practice would be commercially feasible even during a wet prolonged thawing period such as this March has seen.

This was one of those rare years when fall-applied nitrogen was actually utilized during the winter in the Waterville area (Douglas County, Washington); but without the presence of the fungicide, control with the NuGreen alone was completely ineffective. Numerous trials by Holton and later by the writer (1) with various fall-applied fertilizers without added fungicides have not been encouraging. Nitrogen fertilizer, however, is coming into fairly general use in Douglas County and adjacent areas. At wholesale prices the mercurial has been added to the fertilizer at an additional cost of only $2.50 per acre.

In our smaller plots treated by ground application in 1/20-acre units on November 14, 1952, the percentages of green leaf surface left on March 14, March 19, and April 8 were: Agrox (phenyl mercury urea) at 5 lbs. per acre, 15 percent survival; Ceresan M in talc 5 lbs. per acre, 45 percent; PMAS 5 pints per acre, 60 percent; Ceresan M 5 lbs. in 75 lbs. NuGreen per acre, 90 percent; and untreated, 10 percent survival. Both the Agrox and Ceresan M drifted in the slight breeze, and control was spotty in the talc-carried Ceresan M. In 1951-52 where excellent deposit was obtained with a heavier pumicite carrier, Ceresan M without NuGreen gave outstanding control (3).

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STATE COLLEGE OF WASHINGTON, TREE FRUIT EXPERIMENT STATION, WENATCHEE
Project No. 796.

MODE OF INFECTION OF TOMATO PLANTS BY THE ASCOSPORES OF SCLEROTINIA SCLEROTIORUM

Laurence H. Purdy, Jr. and Roy Bardin

Ascosporic infection of tomato plants by the fungus, Sclerotinia sclerotiorum (Lib.) D By., was reported as occurring in the Salinas Valley during the fall of 1950 (1). Symptoms described were whitish lesions over stems and fruits. Pith cavities of infected shoots and stem ends of immature fruits showing lesions contained mycelium and mature sclerotia of the fungus.

In September of 1952, observations were made in a tomato field in the Salinas area in which many plants were infected by this fungus. Most infections observed occurred a foot or more above the soil line and could not have originated directly from sclerotia in the soil. It was concluded, therefore, that these infections also resulted from air-borne ascospores (Figures 1A and 1B).

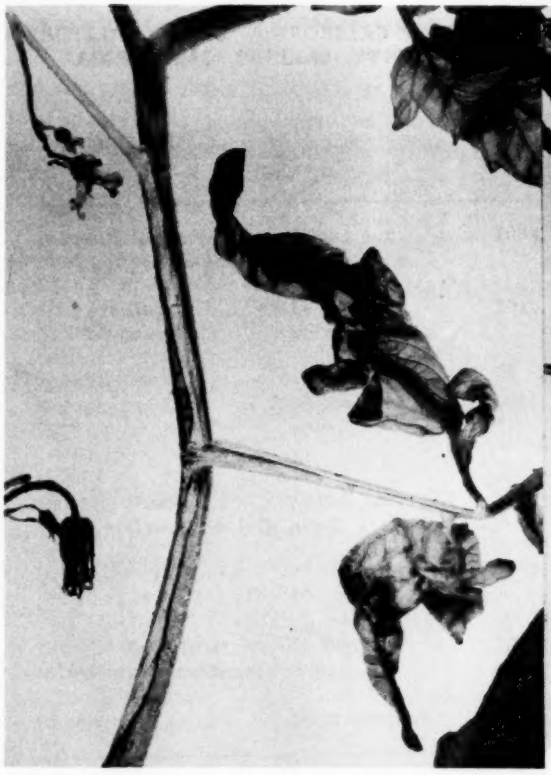

FIGURE 1. A. Lesion produced on tomato shoot by Sclerotinia sclerotiorum resulting from infection of the attached blossoms. Note the green leaflets on the leaf at the right, indicating that infection did not take place in the axil of the leaf.

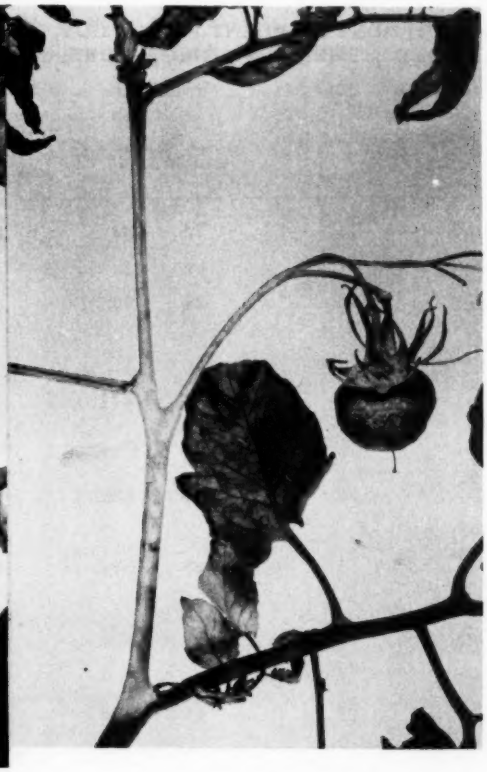

FIGURE 1. B. Infected shoot of tomato with immature fruit still attached showing whitish lesions produced by the fungus. The infection is still active as evidenced by advance of the fungus into the lower leaf axil.

Observations indicated that infection took place through dead flower parts, since in every case the locus of infection could be traced to old flowers which were often still attached to the pedicel. Infected areas were advancing from the base of the peduncle of a fruit spur, and earlier infections were seen in which only the tip of the pedicel was infected.

Inoculations of healthy leaves, and recently opened, senescent, and dead blossoms in a

laboratory moist chamber with a distilled water suspension of ascospores revealed that infection occurred only in the senescent and dead blossoms, since no healthy leaves or recently opened blossoms became infected. However, leaves and flower parts inoculated with a suspension of ascospores in a nutrient solution did become infected.

The conclusions drawn from these field observations and laboratory inoculations are that ascosporic infection of tomato plants under natural conditions does not take place directly through healthy tissue, but only through an intermediary of dead tissue, which in this case was dead flower parts.

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DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, DAVIS, CALIFORNIA AND DEPARTMENT OF AGRICULTURE, MONTEREY COUNTY, SALINAS, CALIFORNIA

PRELIMINARY REPORT ON FUNGICIDE TESTS FOR CONTROL OF DOWNY MILDEW OF CABBAGE SEEDLINGS IN SOUTH GEORGIA

Huey I. Borders

Downy mildew of cabbage, caused by Peronospora parasitica Pers. ex Fr., appears every year in fields of cabbage seedlings grown in South Georgia for shipment to other sections of the United States. Although it is only occasionally that any considerable damage is caused by this disease, the present emphasis on the production of certified disease-free cabbage seedlings has increased the importance of having effective and readily available fungicides for controlling it.

A preliminary report is made of a test conducted at the Georgia Coastal Plain Experiment Station, Tifton, Georgia, during February and March, 1953. These tests will be repeated in the fall of 1953 and again in the late winter and spring of 1954.

Table 1. Cabbage fungicide tests, Tifton, Georgia, February and March, 1953.

Material	Rate of application	Number of plants counted (Total)	Number of diseased plants		Percent of total plants counted that were diseased
			Total	Means	
Spergon Spray (48% active material)	4 lbs. per 100 gallons.	1034	68	17	7
Spergon Dust (4.8% active material)	30 lbs. per acre.	1016	99	25	11
Kolophygon Dust (30% sulfur and 1% Phygon)	30 lbs. per acre.	1000	176	44	18
Parzate Dust (6.5% active material)	30 lbs. per acre.	980	209	52	22
Dithane Z-78 Spray (65% active material)	2 lbs. per 100 gallons.	1035	274	69	28
Dithane Z-78 Dust (6.5% active material)	30 lbs. per acre.	1211	303	76	26
Check	No treatment	983	692	173	70
LSD at 5% level				49	19
LSD at 1% level				67	26

Golden Acre variety of cabbage seed was used in all plots. The sprays were applied at the rate of 150 gallons per acre with a power sprayer at a pressure of 400 pounds per square inch. The dusts were applied with a power duster at the rate of 30 pounds per acre. Treatments were replicated four times in randomized blocks. All plots consisted of 15 plant rows 18 feet long. The rows were spaced 8 inches apart in conformance with commercial practice. Each plot contained approximately 18,000 seedlings.

The effects of the materials tested were consistent throughout, but in order to reduce possible effects of drift from sprays or dusts all counts were made in 3 foot sections of the

five rows in the center of each plot. The results are shown in Table 1.

All treatments reduced the amount of mildew infection but no treatment was greatly superior to the others.

DIVISION OF FRUIT AND VEGETABLE CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AGRICULTURAL RESEARCH ADMINISTRATION, UNITED STATES DEPARTMENT OF AGRICULTURE, IN COOPERATION WITH GEORGIA COASTAL PLAIN EXPERIMENT STATION, THE GEORGIA AGRICULTURAL EXPERIMENT STATION, AND THE GEORGIA DEPARTMENT OF ENTOMOLOGY

LOGANBERRY DECLINE ON VANCOUVER ISLAND[1]

W. E. McKeen[2]

The loganberry plantations on Vancouver Island have exhibited a consistent decline during the past few years and the yield has been much lower than it should have been. The progressive deterioration of many plants in each plantation has not been accompanied by any clearly defined symptoms, but the seriousness of the trouble is now generally recognized and it is evident that an intensive study must be undertaken in order to determine its cause and the remedial measures required for its control. No intensive etiological study has been reported in the literature, but plant pathologists have presumed for some time that the decline is associated with a disease complex.

Loganberry plants appear to develop quite normally during early spring, but after close examination it is found that a reduced number of new shoots emerge from the old crown, particularly in the low-lying areas of the field. Sometimes new shoots begin to emerge from the soil, but suddenly growth stops and they become brown and necrotic. Later in the season when the fruit is beginning to mature, a physiological drought and starvation of the plant seems to occur. The leaves become dry, brittle, curled, and frequently discoloured. The first berries to ripen may be fairly well developed, but the main crop is of inferior quality. The berries are very small and dry, a condition commonly known as "dry berry". The later berries never mature properly, but dry up and turn black on the receptacle. The yield may be reduced by 80 percent.

Although cane gall and crown gall were found to be very serious in many plantings, it was apparent that the tumor-producing bacteria could not be responsible for this serious deterioration, since some plants free from galls were very unthrifty. Also the decline did not appear to be due to a virus because the symptoms were most severe in the low-lying areas.

When the unhealthy plants were removed in mid-summer the crown and root system was not necrotic and new white, healthy roots were present. However, the root system was restricted to a relatively small area about the crown of the plant, and did not appear to be large enough to support the above-ground parts during the fruit maturing period when the weather was dry.

Early in November, 1952, loganberry tips were planted in soil taken from low areas in fields where the decline occurred. The tips started to produce new roots and shoots immediately. However, it was soon observed that in some cases the new shoots did not develop as quickly as expected and some became necrotic before they emerged from the soil (Fig. 3). Examination of the roots showed that a great many were necrotic and frequently were completely decayed.

Tips removed from soil in the field during January and February, 1953, very often were diseased. Usually necrosis began at the very tip of the root (Fig. 1), but it soon involved the whole of it. As these young plants were pulled from the wet soil most of the distal parts of the root system were left behind and only the stubby healthy portions of the roots remained attached to the crown (Fig. 2).

Microscopic examination of many diseased roots showed that a species of Phytophthora was always present and sporangia (Figs. 5, 6, 7, 8, 9) were readily observed about the root. Frequently a species of Pythium was present with Phytophthora, making it difficult to isolate the latter. Occasionally a nematode (Fig. 6) belonging to the genus Paratylenchus was noticed within the necrotic roots. Other organisms were observed sometimes, but at the present time they are not believed to be of any importance. Necrosis of the root systems of old plants was noted, but no isolations were attempted because of the presence of many secondary organisms.

It seemed more probable that the most important pathogens could be found in the newly-formed roots, and the fact that the decline is most prevalent in the low-lying, wet areas of the fields would suggest that water molds are responsible for the decay of the roots.

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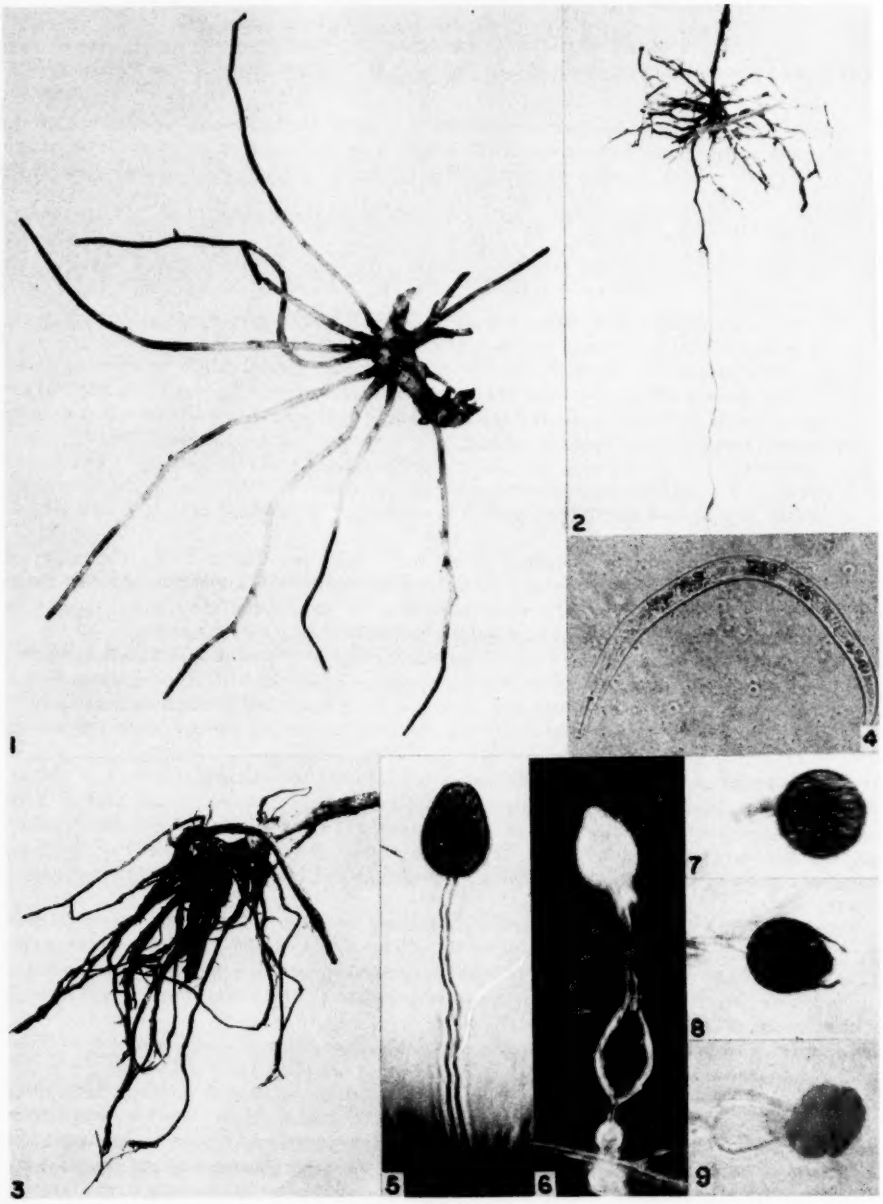
[1]Contribution No. 1278 from the Division of Botany and Plant Pathology, Science Service, Department of Agriculture, Ottawa, Canada.

[2]Acting Officer-in-Charge, Laboratory of Plant Pathology, Saanichton, British Columbia.



FIGURES 1-9. Loganberry decline on Vancouver Island (see page 365).
1 -- A root system of a loganberry tip with most of the roots showing necrosis at the end only. 2 -- A stubby root system resulting from decay of the distal part of the roots. 3 -- A logan tip with a diseased shoot and decayed roots. 4 -- A nematode of the genus Paratylenchus. 5 -- A typical sporangium of the Phytophthora sp. 6 -- A young sporangium forming beyond an old sporangial wall. 7 -- A spherical sporangium. 8 -- A young sporangium within an old sporangial wall. 9 -- Zoospores emerging from a sporangium.

NOTES ON THE FUNGUS CAUSING WILT OF CALOPHYLLUM IN EL SALVADOR, CENTRAL AMERICA[1]

Bowen S. Crandall[2]

In 1949 an epidemic wilt disease of the Central American barillo tree, Calophyllum brasiliense Camb. var. rekoi Santadl., was reported in El Salvador (1). The causal fungus was isolated and tentatively considered to be a species of Cephalosporium. Also in 1949, a wilt disease of C. inophyllum L. var. tacamaha (Willd.) R. E. V. was reported from the island of Mauritius; the causal fungus was described as Haplographium calophylli Wiehe (2).

Information has been received from Dr. M. B. Schol-Schwarz of the Centraalbureau voor Schimmelcultures, Baarn, Netherlands, that comparison of the cultures of the fungus in Central America with cultures in Mauritius shows them to be the same. Behavior of the disease is almost identical in both Mauritius and El Salvador. In Mauritius the disease is thought to have been present since 1904; in El Salvador, only since 1947.

Presence of the disease in Central America has been brought to the attention of officials in the ministries of agriculture in Caribbean, Central, and South American countries, by the Foreign Agricultural Service, United States Department of Agriculture. Forest Service technicians in Puerto Rico supplied seeds of C. calaba and C. inophyllum, since these species are important in reforestation work on that island. Inconclusive inoculation tests on seedlings that grew from these seeds indicated that they were less susceptible to wilt than the native El Salvador variety.

There now appears little question that we are dealing with an example of a tree disease that has come from the Eastern Tropics to the Western. No information, however, is available on how it may have moved from the Indian Ocean area to the Caribbean. The disease in Central America should be considered as being caused by Haplographium calophylli Wiehe.

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COOPERATIVE FIBER COMMISSION, HAVANA

[1]A contribution from the Cooperative Fiber Commission, Havana, a technical agricultural service organization for Cuba, operated jointly by the Government of Cuba and the Foreign Agricultural Service, U. S. Department of Agriculture. United States participation in this work was carried out as part of the Point IV program in Cuba, administered by the Technical Cooperation Administration, U. S. Department of State, through the Institute of Inter-American Affairs.
[2]Mr. Crandall is pathologist, Foreign Agricultural Service, assigned to the cooperative work in Cuba.

CHEMICAL CONTROL OF HYPODERMA LETHALE ON PITCH PINE

C. L. Morris

Pitch pine (Pinus rigida Mill.) is the most widely distributed conifer native to Pennsylvania. Growing in every county, it has attained some commercial importance as a pulpwood and timber producer and has received attention in afforestation of waste banks after coal-stripping operations.

Needle browning of pitch pine is common throughout the natural range of the tree and is especially prominent in plantations. The resulting loss of the food manufacturing ability of needles before the completion of their natural life span is a factor influencing the growth rate and vigor of the affected trees. The importance and possible control of the diseases causing early needle fall prompted this study.

Early browning of the needles of pitch pine in Pennsylvania is commonly caused by several agencies, including a needle-mining insect, Exoteleia pinifoliella, a needle rust fungus, Coleosporium solidaginis, and a needle cast fungus, Hypoderma lethale. Of these, the most important is needle cast or needle drop caused by H. lethale. The fungus Lophodermium pinastri, sometimes associated with needle cast of seedlings of various pines, was found fruiting on numerous occasions on fallen needles beneath trees in plantations and on scattered dead needles remaining on the trees. In most cases the browned needles on the tree had apparently been killed or weakened by attacks of the gall midge or other insects before invasion by Lophodermium.

To study the destructive needle disease caused by H. lethale and to determine if commercial sprays would control the fungus, an experimental area in a 3 1/2-acre pitch pine plantation near Pine Grove Furnace in southeastern Pennsylvania was selected for observation and treatment. Trees in this plantation were 15 years old. This area and the trees selected for spraying were examined at intervals from April 1951 until December 1952.

Infection and Disease Development

During the first week in May 1951 trees infected with H. lethale became conspicuous by the gradual browning of their foliage, which most commonly started at the tips of the needles and progressed towards the base. The browning progressed rapidly and by the middle of May the small shiny black, lens-shaped hysterothecia were developing on the browned portion of the half-killed needles. By June 1, the foliage of heavily infected trees appeared as if it had been lightly scorched by fire, and these browned needles were almost covered with hysterothecia. At this time the new needles were just emerging. A June 8 examination showed the hysterothecia fully developed and containing mature ascospores, although still closed and not releasing any spores.

Three consecutive days of rain during the period June 8 to June 21 apparently were conducive to maximum spore discharge and subsequent infection of the newly emerging needles. By June 21, most of the fruiting bodies had split and few ascospores remained. Some spores are produced and probably discharged throughout the growing season, since mature spores were noted as late as September. However, heavy spore discharge during the period June 8 to June 21 was responsible for the major degree of infection on the new needles as evidenced on analyzing the spray control data (Table 1).

The Spray Control Experiment

Two fungicide spray materials were chosen for comparison of their ability to control needle cast caused by H. lethale. Both were mixed with water, and applied with a standard three-gallon hand pressure garden sprayer equipped with a fine spray nozzle.

Puratized Agricultural Spray, an organic mercurial, was supplied for experimental purposes by the Gallowhur Chemical Corporation. The second fungicide was a fixed copper spray containing 12.75% metallic copper as basic copper sulfate.

Four individual trees were chosen every two weeks for spray treatment. Each tree selected during 1951 had over 50 percent of its needles infected with H. lethale during 1950. Two of the four trees were sprayed with Puratized, each with a different concentration; the remaining two were treated similarly with the fixed copper fungicide. In general, the spray treatment of each selected four-tree group was continued throughout the season as new groups of trees were added to the spray schedule at successive two-week periods. In order to determine the critical period of infection and degree of control during that period, several of the trees selected earliest

Table 1. Results of spray experiments for control of pitch pine needle cast (Hypoderma lethale) Pennsylvania, 1951.

Tree no.	Treatment: material and concentration per 100 gals. water		Period sprayed	No. of sprays	Control on sprayed needles[a]
1	Puratized	1 pt.	May 25 - Aug. 17	7	Excellent[b]
2			June 8 - Aug. 17	6	Good
3			June 21 - Aug. 17	5	Poor
4			July 6 - Aug. 17	4	Poor
5			July 19 - Aug. 17	3	Poor
6	Puratized	2 pts.	May 25 - Aug. 17	7	Excellent
7			June 8 - July 6	3	Good
8			June 8 - June 21	2	Good
9			June 21 - Aug. 17	5	Good
10			July 6 - Aug. 3	3	Poor
11			July 19 - Aug. 17	3	Poor
12	Fixed copper	10 lbs.	May 25 - Aug. 17	7	Good
13			May 25 - Aug. 17	7	Fair
14			June 8 - Aug. 17	6	Fair to poor
15			June 21 - Aug. 17	5	Fair to poor
16			July 6 - Aug. 17	4	Poor
17			July 9 - Aug. 17	3	Poor
18	Fixed copper	16 lbs.	May 25 - Aug. 17	6	Excellent
19			June 8 - July 6	3	Good
20			June 21 only	1	Fair
21			July 6 - Aug. 3	3	Poor
22			July 19 - Aug. 17	3	Poor
23	Check	none			Over 60% of needles on all check trees were infected with needle cast during 1951 spray period
24					
25					

[a] Based on observations April to November 1952.

[b] Excellent=less than 1% of sprayed needles with cast infection; good=1%-5%; fair=5%-15%; poor=over 15%. Percentage of infection was an estimate based on sampling of needles on four or five branches scattered throughout the crown of the sample trees.

were dropped from the spray schedule at various periods. Trees selected for spray treatment averaged 6 feet in height and required about 1 1/2 quarts of spray solution for each treatment.

To increase the sticking properties of the spray, 5 cc. of the U. S. Rubber Company's experimental fungicide sticker were added per gallon of solution of Puratized Agricultural Spray, and the same proportion of potassium oleate, a soap, was added to the fixed copper spray.

The experimental sticker was of little advantage since most of the latex it contained precipitated on addition to the spray solution. This separation may have been due to low temperatures to which the sticker was subjected on several occasions before use.

Visible fungicide residue from the copper spray on needles sprayed two weeks earlier was often noted. The residual effect of Puratized, a colorless liquid, was difficult to gauge, but the excellent control obtained (Table 1) indicated that this property is good.

Heavy infection was noted on needles produced during 1950 on trees in the test area and examination of check trees indicated that the intensity of infection was equally heavy during the following year when spray tests were carried out. The same conditions applied to several other pitch pine plantations and to naturally seeded pines in other areas under casual observation during this period. In 1952, however, infection was considerably lighter. In the study area, there is some indication that the trees effectively protected by spray in 1951 were less heavily infected in 1952 than their unsprayed neighbors.

Observations extending over the two-year period 1951-1952 indicate that it is difficult to predict the degree of infection to be expected from H. lethale. Infection by this needle cast fungus apparently depends on favorable moisture and temperature conditions at a critical period in the early growing season when spores are mature and the newly developing needles are tender and susceptible to infection. Total monthly rainfall during the critical infection period (June in 1951) apparently was not a criterion.

Results of the Spray Control Tests

(1) Two fungicides tested under field conditions gave practically complete control of the needle cast fungus Hypoderma lethale on pitch pine.

(2) Both Puratized Agricultural Spray at the rate of one pint and two pints per hundred gallons of water and a fixed copper spray at the rate of 16 pounds per hundred gallons of water gave excellent control when applied at two week intervals throughout the growing season.

(3) Although some infection of newly emerging needles by H. lethale apparently takes place throughout the growing season, observations during 1951 indicate that heaviest infection takes place within a relatively short period in late spring (the middle two weeks in June). It is possible to obtain good control of the fungus by a few applications of a fungicide during this critical infection period. Puratized Agricultural Spray at the rate of two pints per hundred gallons of water during this period of heavy infection gave much better control of the fungus than did the fixed copper spray applied during the same period.

(4) Foliage on trees where infection by H. lethale had been appreciably reduced by spraying was retained an average of eight months longer than foliage on the same trees which had been heavily infected with this needle cast fungus the year before.

(5) The fixed copper spray when applied at the rate of 16 pounds per hundred gallons of water throughout the growing season caused appreciable stunting of the newly emerging needles. This stunting did not seem to affect seriously the general vigor of the sprayed trees, however. Unsprayed needles which emerged the following year on the same trees were normal in length.

(6) Observations in the sprayed plantation indicate that, generally speaking, the more vigorous trees were less heavily infected with the needle cast fungi. All trees under study in the plantation showed heaviest cast infection on needles in the lower crown. These conditions may be associated directly or indirectly with vigor, with the method of spore dispersal and with the air movement and comparative rates of drying at different levels above the ground.

DIVISION OF RESEARCH, PENNSYLVANIA STATE DEPARTMENT OF FORESTS AND WATERS

ADDITIONAL RECORDS OF SPOT ANTHRACNOSES IN MARYLAND[1]

R. A. Jehle, Anna E. Jenkins, and W. F. Jeffers[2]

Our résumé of eleven different spot anthracnoses that by 1950 had been found in Maryland (4) has since been supplemented by additional records of violet scab (1) and flowering dogwood spot anthracnose (2, 3, 5).

A number of specimens still unreported increase the known distribution in the State of five more of these eleven diseases. These specimens are tabulated here in order that the new data they afford may be available for reference (Table 1).

English ivy scab and snowball anthracnose have previously been reported in Maryland only from the Eastern Shore: ivy scab from Salisbury, Wicomico County, 1949 and 1950, (4, tab. 1 and fig. 1, D); snowball anthracnose from Marion, Somerset County, and Pocomoke, Worcester County (6).

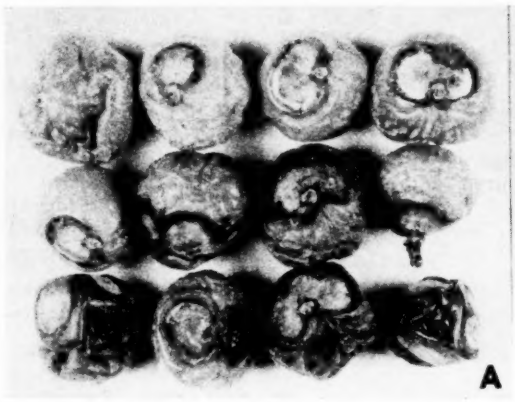

FIGURE 1. (A) Grape anthracnose caused by Elsinoë ampelina collected July, 1952 from Pocomoke, Maryland.

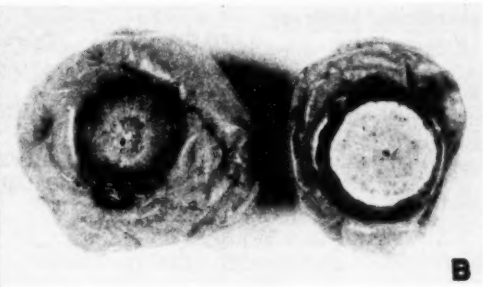

FIGURE 1 (B) Close-up of two infected berries.

In its earlier history in the State, grape anthracnose seems to have been recorded only as rare or mild in its attack. Last year, however, it was unquestionably severe on berries at Pocomoke (Fig. 1). From horticulturists at Beltsville we learn, too, that the disease is sufficiently common to be troublesome in their experimental plots.

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2. _____ and _____. 1952. Northernmost records of flowering dogwood spot anthracnose in Maryland. Plant Dis. Reptr. 36: 110.
3. _____ and _____. 1952. Known distribution of flowering dogwood spot anthracnose in Maryland, May 1952. Plant Dis. Reptr. 36: 296-297.
4. _____, _____, and W. F. Jeffers. 1951. Spot anthracnoses known in Maryland and their distribution by counties. Plant Dis. Reptr. 35: 194-199.
5. Jenkins, A. E. 1952. Spot anthracnose of flowering dogwood found in the Nassawanda Forest, Maryland, and isolation of the pathogen. Plant Dis. Reptr. 36: 294.

[1]Scientific article No. A424. Contribution No. 2460 of the Maryland Agricultural Experiment Station. Department of Botany, University of Maryland, and U. S. Foreign Agricultural Service, Instituto Biológico, São Paulo, Brazil.
[2]We are indebted to Dr. A. A. Bitancourt, Instituto Biológico, São Paulo, Brazil for his collaboration in diagnosis of the spot anthracnoses of English ivy and wild grape reported here.

Table 1. Previously unreported collections of spot anthracnose in Maryland.

Disease, cause and host	Locality: County	Locality: Town	Date of collection	Collector	Remarks
Bramble anthracnose (Elsinoë veneta)					
Rubus sp. (wild)	*Garrett	Friendsville vic.	6-29-50	R. A. J.	Rare
R. sp. (wild blackberry)		Oakland vic.	6-20-51	R. A. J.	Common
English ivy scab (Sphaceloma hederae)					
Hedera helix	*Baltimore	Towson	1-15-47	R. A. J.	Nursery; rare
			7-10-51	M. A. Clough	Garden; abundant
		Baltimore	10-13-51	Mrs. F. Hartig	Garden; abundant
		Elkridge	3-28-48	R. A. J.	Rare
	Wicomico	Salisbury	3-12-51	R. A. J.	Garden; moderate
			9- 9-51	R. A. J. and A. E. J.	Same garden; abundant
Grape anthracnose (E. ampelina)					
Vitis sp. (wild)	*Garrett	Grantsville vic.	7-13-51	R. A. J.	In woods; rare
V. sp. (wild x V. vinifera parentage)	Prince Georges	Beltsville	6-11-51	R. A. J. and D. H. Scott	Experimental plot; U.S.D.A. Plant Industry Station
Vitis sp. (cult.)	*Worcester	Pocomoke	7-30-52	W. F. J	On berries; severe
Rose anthracnose (S. rosarum)					
Rosa sp. (cult.)	*Garrett	Eagle Rock	6-25-50	R. A. J.	Abundant
		Swanton	6-20-50	R. A. J.	Abundant
R. multiflora	Prince Georges	College Park	6- 8 to 10- 2-51	R. A. J.	Campus, U. of Md.; rare in June, abundant in October
Snowball anthracnose (S. viburni)					
Viburnum opulus var. roseum	Prince Georges	Clinton	9-14-51	R. A. J.	Abundant

* Counties from which no definite record of the disease concerned was previously available.

6. Jenkins, A. E. and R. A. Jehle. 1950. Sphaceloma viburni destructive to snowball in Maryland. Plant Dis. Reptr. 34: 350-351.

UNIVERSITY OF MARYLAND, AND U. S. FOREIGN AGRICULTURAL SERVICE

UNUSUAL RECORDS OF PLANT DISEASE OCCURRENCE

SIDE CATFACES IN TOMATO FRUITS IN TEXAS

By P. A. Young

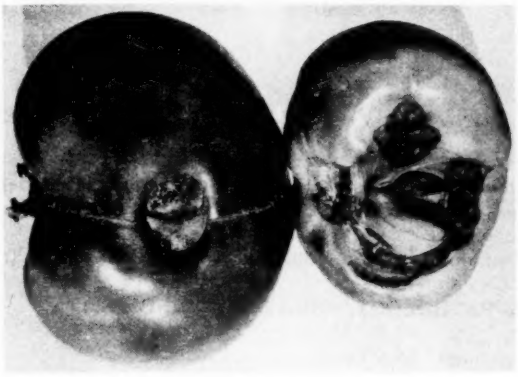

FIGURE 1. Tomato fruits with different types of side catfaces. On the ripe fruit on left the catface is in the center of a vertebrae-crack line. The green fruit on right shows deep cracks that broadened as they healed.

Catfaces in the sides of tomato fruits were unusually abundant in June 1952. They consisted of healed cracks or holes 3 to 30 mm. wide and as deep as 10 mm., often with curved smooth sides (Fig. 1). The deepest catfaces exposed the seeds. Many side catfaces had vertebrae-lines attached to them, and apparently developed by the enlargement of such crack lines. In certain cases the lines broadened to form wide, peel-deep, corky meridian bands. Side catfaces apparently started developing when the fruits were very small. Abnormal adhesion of the corolla was a possible cause. Ordinary catface symptoms occur in the blossom end of fruits and vary widely in appearance. The shallow line-type of catface is a vertebrae-crack line across the blossom end of a tomato fruit[1].

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[1]Young, P. A. Tomato diseases in Texas. Texas Agr. Exp. Sta. Circ. 113. 1946.

POOR CONDITION OF ALFALFA IN VIRGINIA FIELDS

By S. B. Fenne

Many complaints have been received from county agents and farmers in Virginia concerning the poor growth and yellowed condition of alfalfa seeded last fall. These yellowed plants showed blackstem and two or three leafspot diseases; however, the yellowing could not be explained on the basis of these diseases alone. An examination of the roots of the yellowed plants failed to show any nodules present. In certain portions of most fields, vigorous green plants were found which showed an abundance of nodules.

Most of the farmers prepared the soil well for seeding and carefully followed the recommendations of the College in regard to fertilization, inoculation, etc. However, last fall was unusually dry and apparently the inoculum died out before it could become established on the young alfalfa plants. In certain low portions of the field or in tire tracks following the tractor, apparently there was sufficient moisture present to provide protection against drying out and under such conditions the alfalfa looked normal and the roots of the plants showed good nodulation. It appears, therefore, that the soil last fall at seeding time was too dry, in some sections of Virginia, for the alfalfa inoculum to survive.

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RHIZOCTONIA BLIGHT OF FITTONIA

By C. W. Ellett

Species of Fittonia, a genus of Acanthaceae native to South America, are cultivated in greenhouses as foliage plants. In 1952 in greenhouses in Ohio, a leaf and stem blight was observed on Fittonia verschaffeltii var. argyroneura. Symptoms on the leaf blades occur as watersoaked or scalded areas and lesions on the petioles and stems as a soft decay of the tissues. Rhizoctonia solani was isolated from the affected parts and when healthy plants were inoculated, symp-

toms developed similar to those described above.

Fittonia is not listed in the Index of Plant Diseases in the United States, Part 1 issued in July, 1950 as a Plant Disease Survey Special Publication, and a check of the readily available literature does not reveal any reports of this disease.

DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, OHIO STATE UNIVERSITY, COLUMBUS

RHIZOME ROT OF GERANIUM

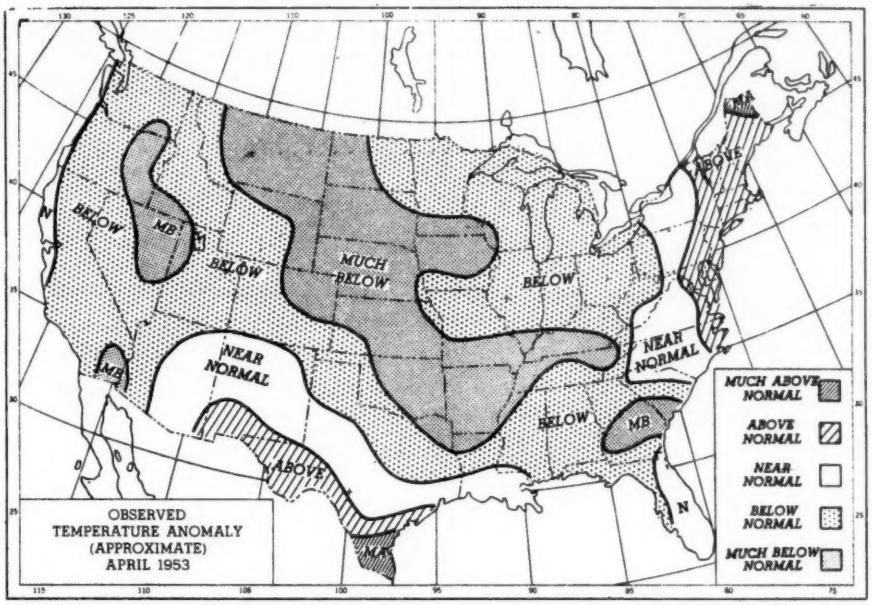
By C. W. Ellett

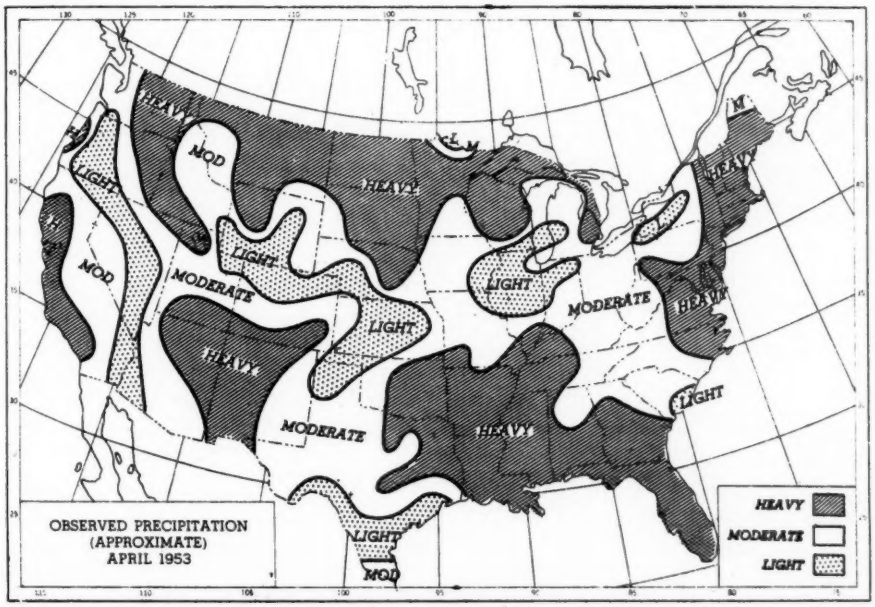
In April, 1953 in Madison County, Ohio, the apothecia of Seaverinia geranii were observed growing from partially decayed rhizomes of Geranium maculatum. The Botrytis conidial stage was also found on the rhizomes. Cultures were obtained both from ascospores and from conidia and the Botrytis stage developed in these cultures.

This is the first report of this fungus and disease in Ohio. Seaver[1] records the fungus from New York and Wisconsin.

DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, OHIO STATE UNIVERSITY, COLUMBUS

[1]Seaver, F. J., 1951. The North American cup-fungi. New York.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.